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—EDITOR.

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EDITORIAL

IF necessity is the mother of invention, it must have been true in 1897, for Keegan records in his treatise entitled "Rhinoplastic Operations" that in that year alone, 152 rhinoplastic operations had been performed in India and that included in the series were one that went to the credit of the Madras Hospitals and three to the rest of the Madras Presidency!

The question posed is where was the necessity for so many such operations in our country then? The answer seems simple. Too many people appear to have lost their noses and required reparative therapy to cover their ugliness. It is strange that the etiology of the mutilation was not 'accidents' to the nose; rather, ablation of noses was a punishment meted out for sexual crimes. It is authentically stated that in Nepal, then, cutting off the nose was an approved punitive measure finding a place in the statute book.

If rhinoplastic surgery to-day were relegated to the limbo of forgotten practices and is not what it had been then, can we take it that sexual crimes have gone down to such an extent or that the mode of punishment for such delinquencies has completely altered? Whatever the answer, in the field of Venereology, the occasional patient, there is, that reports with such an anatomical loss, even to-day, due to neglected or mal-treated venereal disease.

Common amongst the venereal diseases credited with such disastrous outcome are :

1. Treponematoses which may be

    (*a*) Venereal, acquired or congenital.

    (*b*) Non-Venereal, acquired syphilis in childhood.

    (*c*) Non-Venereal Treponematosis under the appellation Yaws, with special category—*Gangosa.*

2. Donovanosis, the other venereal disease entity, aside from the facial destruction, terminates with or without treatment, in a few instances, with stenosis of the vantage points in the male urethra, prepuce, cervix, vagina, anus and sometimes the mouth.

3. Lymphogranuloma Venereum results in the formation of unusual channels like vesico-vaginal fistulae, recto-vaginal fistulae and the effluvia then have to pass *via* abnormal routes.

V. D. patients also present themselves with concomitant non-venereal conditions :

1. Fusospirillosis which similarly destroys parts of the anatomy.

2. Hansen's and/or Koch's infections produce similar destructive facial lesions.

The victims of venereal diseases with such disastrous consequences contrive to camouflage their defects very skilfully and conduct themselves in society with great complacence. It seems as if they make no attempt to get their deformity corrected. While the patients' attitude may thus be described as callous, the physicians appear to be in a predicament. The medical attendants have been constrained to merely prescribe to combat the infections. Probably, nothing is amiss in this act of ours. The administration of specific remedies appropriate for the clinical conditions as per the standard schedules certainly cover

the infectious agents. But all of us realise that none of these chemotherapeutics or antibiotics at our disposal can take on the onerous duties of repairing the damaged human anatomy. The surface lesions heal but the disfiguring scars are left behind permanently. It is not anatomical defects or deformities alone but the consequent physiological dysfunctions which perturb the patients, physicians and the public. The patients indeed suffer from a therapeutic paradox—a cure with an inability to carry out acts like micturition, defaecation, menstruation, parturition and sexual congress or even eat food. The mental agony of these patients has to be witnessed to be appreciated.

Plastic surgery in venereology is a desideratum despite the fact that our demand for the services of a plastic surgeon from the venereologists' angle may be disproportionate to the number of venereal cases with deformities requiring relief.

In this context, the visit of Sir Harold Gillies has heralded the hope that the subject of plastic surgery will be reviewed, renewed, revived and given the first priority. The plastic surgeon's masterly presentation through projection of pictures of his successes, has been a great eye opener to us in the Madras Medical Service. Now that the necessity for plastic surgery has risen, may we hope that our mother country will invent plastic surgeons soon, so, as in the past!

P. N. Rangiah.

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MADRAS

Cirrhosis of Liver

Dr. R. SUBRAMANIAM*
B.SC., M.D., M.R.C.P. (LOND.)

The problem of Cirrhosis Liver has not ceased with the introduction of Prohibition. That shows alcohol is not *the* factor though it may be one of the many that may cause cirrhosis liver. The incidence of cirrhosis in the Government General Hospital, Madras, for the last seven years is given below. The table also shows the incidence in my wards. From this may be gathered the high rate of incidence of this disease in this part of the country. It continues to be an important clinical problem in South India.

Year.	*Total No. for General Hospital.*	*Dr. R. Subramaniam's Unit.*
1952	284	79
1953	296	60
1954	375	44
1955	444	68
1956	446	85
1957	238	75
1958	424	86

The name 'Cirrhosis' was given to the condition by Laennec to indicate the tawny colour, but to-day it is not only the colour that is responsible for the name but it is the fibrotic change that occurs in the liver that makes the clinician as well as the pathologist call this as Cirrhosis Liver. The name 'Cirrhosis' is very inappropriate in that the word cirrhosis does not mean fibrosis. It is called Laennec's cirrhosis, though it was

*Professor of Medicine, Madras Medical College, Physician, Government General Hospital, Madras.

not Laennec who described the condition. Laennec described it long after others described it. It is also called Atrophic Cirrhosis. In the early stages at any rate, there is no atrophy. It is called alcoholic cirrhosis, but the condition is known to occur in perfect tee-totallers. It is called Gin-Drinkers' liver, and the objection that I place on alcohol will apply with equal force to gin as well. So the name is unsatisfactory, though every one is able to understand what one is talking about.

The Etiology of Cirrhosis Liver.—As medical students you were taught alcohol is an important problem, but to-day we see a good number of cases occurring in non-alcoholics. Even as early as in the beginning of this century, there were clinicians who felt that alcohol may not be the sole factor, though they were not able to pin down the etiological factor at that time. To-day our view about the etiological factor of cirrhosis liver is that it is largely due to Infective Hepatitis occurring in mal-nourished individuals. The infective hepatitis itself need not necessarily be accompanied by jaundice. When jaundice is absent, the infective hepatitis may be missed. When infective hepatitis occurs in a mal-nourished individual and he is taking first-class proteins, the chances are he may die of the attack of infective hepatitis, but if he survives, the recovery is so good that no residual effect is seen. In the mal-nourished person, infective hepatitis is not accompanied by high mortality at the initial stage. Several factors may be responsible for cirrhosis liver. They may all be acting synergistically or in succession one after the other with the result that the liver has no chance of completely recovering or asserting itself. The most important cause in a mild series of cases was the lack of sufficient intake of first-class proteins. By first-class proteins, I mean proteins of animal origin. In the large majority of persons, they were taking diets which were mainly made up of boiled rice, skimmed buttermilk diluted, and one or two chillies. The protein value of such a diet is extremely low. Though the protein value is low, they are able to go through without symptoms, so long as they are free from any other infection. If they contract any infection, it ordinarily may not produce any adverse results, but these patients tend to get

cirrhosis liver. It looks as though their liver can just manage to survive, and this extra load of infection causes the liver to go into a state of failure.

The common infections which favour the incidence of cirrhosis liver are Amoebiasis, Kala-azar and Malaria. Fortunately Malaria has been brought under control and we no longer see the recurring attacks of malaria that was such a common feature ten years ago. The liver damage that may occur as a result of Kala-azar is not so great as compared to damage that may occur due to the antimony that is used in the treatment of Kala-azar. Fortunately the ascites that occurs as a result of antimony therapy is fairly quickly relieved, since the metal is not used repeatedly. As regards amoebiasis, it is the alcoholic with amoebiasis that suffers from liver complications rather than the non-alcoholics. Even so, it is worthwhile examining the motions of all cirrhotic patients, even of non-alcoholics 3 or 4 times and at least once after the saline purgative for evidence of amoebic cysts. If amoebic infection is present, treatment for the same gives very gratifying results.

Presence of ankylostome infection worsens the cirrhosis. Here the ankylostome infection causes anaemia and fatty degeneration. In cirrhosis liver there is already a tendency for fatty degeneration. So the burden of damaged organ is lessened by de-worming. In patients whose liver is in a damaged state, it is preferable not to de-worm the patients straight off, but to give protection to the liver by giving calcium and glucose, preferably parenterally, for a few days and then de-worm the patients.

Though not a common factor, Syphilis should always be borne in mind. In the course of a year or two years, we come across a case of ascites in whom the serological reaction is positive in very high dilutions. In my opinion, one should not wait for a post-mortem examination to confirm the syphilitic etiology of cirrhosis liver. Just as we diagnose syphilitic mes-aortitis when we come across a dilated aorta proved either by screening or by X-rays, coupled with chest pain and a positive serology

and evidence of syphilis elsewhere as shown by enlarged supra-trochlear glands or posterior cervical adenitis or syphilitic leukomelanodermia. In these cases the syphilitic etiology of cirrhosis is further confirmed by the dramatic response to anti-syphilitic treatment.

An important associated disease that occurs with cirrhosis liver is tuberculosis. Tuberculosis may occur in the lungs in all cases of cirrhosis liver and as a prominent manifestation. Tuberculosis of the lungs and peritoneum may also occur. These two conditions are generally fairly quickly spotted by the alert clinician, but when tubercular peritonitis occurs alone, the positive data are so meagre that one does not think of the possibility of tuberculosis. The possibility of tubercular peritonitis should be visualised when the patient is admitted with fever. The fever itself is irregular in character and irregular lumps may be felt in the abdomen. The lumps themselves are not evident like the intra-abdominal organs like spleen, kidney or liver. They are comparatively mobile, with the result that they are not constantly felt, or even if felt, they get easily displaced. The temperature that may occur with uncomplicated cirrhosis is usually never high, generally of the order of 99° or 100°, but when tubercular peritonitis occurs, it may go up to 101° or 102°, and there is well-marked loss of appetite. The loss of appetite is not peculiar to tubercular peritonitis when it occurs in the course of many other conditions like carcinoma of the stomach or kidney disorders. Once the possibility of tubercular peritonitis is visualised, the diagnosis can be confirmed by aspirating the ascitic fluid and having it biochemically analysed. Serum protein content exceeding 3 grams, the presence of lymphocytes, coupled with well-marked Mantoux reaction is highly suggestive of tubercular peritonitis. With the introduction of Prohibition, alcohol has ceased to be an important problem. Even prior to that, it is only in 40% of patients, we used to elicit a history of alcoholism. It is only when alcohol is taken by those whose diet is deficient in first class proteins from which the body can derive essential sulphur containing amino-acids like methionine and cystine, cirrhosis used to occur. Where the diet has been good,

intake of alcohol, so long as it has been strictly moderate, has never been an important factor in the causation of cirrhosis liver. We have seen in our wards that patients suffering from hypovitaminosis, particularly A, B complex, and choline, are likely to develop cirrhosis liver. Where patients complained of night blindness, liver biopsy studies showed pictures identical with early cirrhosis, *i.e.* fatty degeneration. Among the various factors I have listed, nutritional deficiency is the most important, particularly lack of essential sulphur containing amino-acids like methionine and cystine, and also hypo-vitaminosis. The other factors that I have listed seem to precipitate rather than act by themselves in the production of cirrhosis liver.

PATHOLOGY

In the early stages the liver is enlarged, and liver biopsy at this stage shows fatty infiltration. The fatty infiltration is seen in the histo-pathology slides, as clear vacuoles. Even at this stage, prominence of the fibrous barrier is seen. In the autopsy room, the liver is generally small in size. Hence the name atrophic cirrhosis liver. Rarely it may be normal or rather more than normal in size. Bulk for bulk, the cirrhotic liver is heavier than the normal liver. It is the congestion and fatty infiltration that make the liver larger in size. In younger subjects the liver is large in size. In non-alcoholic cirrhosis, the liver is usually small. The large livers have a fine morocco leather-grain appearance, for the naked eye. The small livers generally have a rough surface, which is coarsely nodular. Hence the name 'Hob-nail liver'. The projecting nodules are the re-generating or hypertrophied liver cells and the depressions in between are due to the fibrous tissue. In the literature, the average weight of the liver is described as 52 ozs. In Madras, in the General Hospital series, the weight was only 35 ozs. A study for 30 years from 1928-58 revealed the minimum weight to be 16 ozs. and the maximum weight to be 134 ozs. The liver may be adherent to the neighbouring structures particularly to the diaphragm, or to the viscera like stomach, duodenum or colon. The nodules though usually about ½" in diameter,

occasionally reach the diameter of a well-developed hen's egg. The hob-nails themselves never show umbilication in contrast to secondary deposits. The changes that occur in the liver as a result of cirrhosis are uniform. Since usually no part of the liver escapes, liver biopsy gives a very good indication of changes that are taking place in the liver at the time of the biopsy. There is no need to doubt whether the biopsy will represent a correct perception of what is happening in the liver. Unlike malignancies in the liver, primary or secondary where changes are likely to be confined to select areas in the liver, cirrhosis is a uniform process. Though the process is uniform, the left lobe is more affected than the right, but in no case have I come across any particular lobe being completely spared. The reason why the left lobe is more affected may be due to the fact that most of the blood from the small intestines goes to the right side of the liver, and so nutrition is extracted by the right side, whereas blood from the spleen or the colon is likely to go to the left side that is blood devoid of nutritive material. The lack of nourishment occurring in a de-vitalised state may cause atrophy of the left lobe. The liver on section is tough and presents the same appearance as on the surface. The appearance is like that of a mosaic, that is a number of stones embedded in a uniform base. The liver substance itself is pale in colour in contrast to the healthy liver, and has an yellowish brown colour due to fatty changes with bile-staining. There may be areas of haemorrhage in the interstitial tissue cells.

HISTOLOGY

In the early phase, there is small cell infiltration around the portal spaces. The cells are due to hyperplasia of the existing connective tissue of the Glisson's capsules including the Kupffer cells. In advanced cases there is an irregular mass of connective tissue extending throughout the liver and dividing it into varying islets of liver cells. A number of lobules are enclosed in the frame work of the connective tissue—the number in each frame work varies widely. In the early phase the liver cells show fatty infiltration and in the late phase the cells undergo cloudy swelling, become granular and the nuclei stain

badly. There may be evidence of active division as shown by mitosis, particularly in areas of hyperplasia. In the late cases, the normal pattern of the liver is completely lost. The distortion is particularly well-marked with regard to the vascular pattern and anastomoses between the branches of portal and hepatic veins occur.

Clinical features.—The clinical features of cirrhosis liver can be divided into two stages, namely the compensated stage and the de-compensated stage or, the ascitis-free stage and the ascitic stage. In the compensated stage, the symptoms are vague and indefinite. The symptoms experienced may be night blindness or endocrine changes. In the case of men, loss of libido and impotency, loss of hair particularly over the chest and axilla and also on the face, (so that the necessity for shaving becomes less urgent) and the tendency for feminine type of distribution of pubic hair are commoner. In the case of women, amenorrhoea or metropathia haemorrhagica may occur. The symptoms in the compensated stage may be loss of appetite or inability to digest food as well as previously ; or the quantity consumed becomes progressively less with the result that weight loss occurs. Or, these patients may complain of not being able to do the day's job without getting unduly tired. Sometimes they complain of heaviness or fullness after meals. Even though they are free from ascites, haematemesis may occur. As the symptoms are vague at this stage and are non-specific in character, the possibility of cirrhosis is not thought of as a rule, and is likely to be missed unless one is cirrhosis-liver conscious. In a small percentage of cases hypertension has been recorded at this stage of the illness and the pressure falling down without any medication as the disease advances. Clinical examination at this time reveals moderate degree of hypochromic microcytic or normochromic normocytic anaemia. The liver itself is palpable 1 to 1½ inches below the costal margin with a smooth surface, and it is neither tender nor hard. The bio-chemical tests are essentially normal at this stage. One can only arrive at the diagnosis of cirrhosis liver by doing a liver biopsy.

The liver biopsy is a *must* in cases of suspected cirrhosis liver. If one is to diagnose cases of cirrhosis liver in the early stage, a stage at which complete correction is possible, it can be done only by doing liver biopsy. The liver biopsy is a comparatively safe and easy procedure, provided certain precautions are observed. In my wards, we do a little over 100 biopsies in a year. Before doing a liver biopsy, we give our patients 10 c.c. of 10% calcium gluconate with 1 c.c. (10 mgms.) of water soluble Vitamin-K, intravenously daily for ten days. On the 3rd day, about an hour before the injection, the liver biopsy is done. Usually we prefer the 8th or 9th right axillary line, but where there are nodules to be felt through the anterior abdominal wall, then liver biopsy is attempted *via* the anterior abdominal wall. I do not recommend this as a routine procedure particularly in inexperienced hands. The liver biopsy in the compensated stage shows fatty degeneration of the liver, when there is evidence of fibrosis, fibrosis itself being minimum but more than normal. The liver retains its normal architecture, but there may be areas of focal necrosis with collections of inflammatory cells indicating the liver damage that is going on. Clear round spaces are seen predominantly in the peri-lobular liver cells. These are due to the fatty degeneration occurring in the liver. With present-day standards of treatment if spotted at this stage of the illness, we should be able to completely clear the condition and restore the patients to normal health. If not spotted at the early or compensated stage, and if no treatment is given, in about 6 months to 12 months time from the onset of the first symptoms, the patient shifts to the de-compensated or the ascitic stage.

The de-compensated stage is comparatively easy to diagnose; since the patient presents himself with ascites. Ascites is the sole clinical feature in the early phase of decompensation, but as the disease advances, oedema of the legs also occurs. Symptoms at this stage may be due to the liver cell damage, *i.e.* parenchymal ...... or due to portal hypertension. Symptoms secondary to liver cell damage are (1) ascites, (2) ankle oedema, (3) jaundice,

(4) loss of libido, partly due to testicular atrophy, (5) gynaecomastia, (6) alopecia-pectoral, axillary and facial, (the patient's need for a daily shave is not urgent, and later on there is no need for a shave at all). (7) spider naevi, (8) palmar erythema, (9) anaemia-microcytic rarely it may be macrocytic, (10) haemorrhagic tendencies and (11) coma.

The symptoms due to portal hypertension are mainly to be seen in the blood vessels particularly at the junction of the systemic and the portal vessels. So the early symptoms may be (1) haematemesis, (2) splenomegaly, (3) caput medusae, that is, prominence of the veins around the umbilicus—the prominence of the veins may be more in the case of inferior vena cava—(4) ascites, (5) anaemia and (6) ankle oedema. Portal hypertension may also cause bleeding piles and although piles can occur in many conditions other than cirrhosis liver, it may be considered as an important symptom of portal hypertension.

Diagnosis is arrived at by the gross oedema below the xiphisternum and the wasting above the xiphisternum. The characteristic hepatic facies, namely the sunken eyes and hollow cheeks, is a late manifestation, and the clinician who wants to cure should not wait for the typical facies to occur.

Another important feature particularly in advanced cirrhosis liver is that the patients are able to attend to their personal needs themselves in spite of well-marked ascites. If untreated or only treated symptomatically, deterioration of the clinical condition and death takes place in 6 to 8 months' time. With the onset of ascites, demonstrable biochemical changes occur which help to diagnose cirrhosis liver. An important change is the lowering of the protein content and of albumin in particular, in the elevation of globulin content and increased thymol turbidity. The portal hypertension can be demonstrated by means of portal venography done by injecting diodrast or any other suitable contrast media into the spleen. This helps to visualize the splenic vein and also gives a clue to the site of obstruction. This procedure is particularly useful where surgery

is contemplated. In one of my cases with a grossly enlarged spleen and ascites, portal venography showed block in the splenic vein itself indicating the obstruction was before the formation of the portal vein. Evidently it is a case of splenic vein thrombosis. The usual course is for the obstructed portal venous channels to pass through the lower end of oesophagus and from there join the inferior vena cava above the dome of the diaphragm.

Differential diagnosis has to be made from ascites as part of general anasarca, as it occurs in cardiac conditions, renal conditions, nutritional oedema and advanced anaemia. The clinical examination eliminates the cardiac condition, and examination of the urine will eliminate renal causes. General inspection will reveal whether the patient is anaemic or not. The condition has got to be distinguished from other local causes of ascites like tubercular peritonitis, Banti's disease, primary or secondary malignancy of the peritoneum; obstruction at the hilum of the liver may cause ascites and jaundice. Obstruction of the thoracic duct results in chylous ascites. It is not possible to say clinically that one is dealing with chylous ascites, straight-off. The usual cause of chylous ascites is filariasis, but in western countries it is due to malignant disease invading the thoracic duct. Thrombosis of the inferior vena cava can also cause ascites. Here the superficial veins of the abdomen are more grossly prominent, more in front than in the back. The thrombosis in this case is above the renal veins. The obstruction of the inferior vena cava can also be demonstrated by dye injection in the lower limbs.

TREATMENT

Having established the diagnosis of cirrhosis liver, the question of treatment arises. As in all diseases, prophylaxis is the best. The principle is to prevent the factors from operating and causing the disease. This being so, the nutrition of the entire population has to be made up, so that lack of essential amino acids may not occur in the diet. With this end in view, people

should be encouraged to take milk, preferably from cows which are not stall-fed. They should also include protein of animal origin like mutton, egg and fish. The quantity of milk may be increased in the case of vegetarians. The various preventable maladies like amoebiasis, ankylostomiasis and dysentery should be prevented. In the compensated stage, a high degree of suspicion is necessary to spot the case and the disease can be confirmed by doing a liver biopsy and treated by liberalising the diet including essential amino acids. In the de-compensated stage, the essential thing is to give relief to the patient. As often as he complains, paracentesis abdominis should be done, so that the patient is not in distress. Unduly long delay in doing paracentesis is not desirable. Unnecessary drainage is also not desirable, because the fluid contains proteins which are very valuable for the body metabolism, and also by frequent paracentesis collateral drainage channels are not likely to be developed. The need for frequent paracentesis can be avoided by putting the patient on mercurial diuretics like salyrgan, one c.c. intramuscularly, not oftener than once a week. It is also not advisable to put the patient on ammonium chloride, as it may precipitate hepatic coma. A diuretic like Diamox is also contra-indicated in the treatment of cirrhosis liver. While there is not the same objection to the use of chlorothiazide liberal use of this drug is not advisable but only small doses during times of urgent necessity only.

It is of great advantage to have bed rest in the de-compensated stage as it brings down the work of the heart and enables the patient to recover quicker. If the decompensation is not too severe, he may be given Methionine 1/2 gm. t.d.s. p.c. The recommended dose for western patients is 1½ gms. 3 times after food. This large dose in the South Indian subject is likely to cause severe diarrhoea. Liver extract can also be given with great advantage. In my wards we use 1/2 c.c. as test dose, and if no untoward reaction is noticed, 1 c.c. in 20 c.c. of 25% glucose solution is given intravenously. Each day the dose of the liver extract is increased by 1 c.c. maintaining the proportion

with the glucose until we reach 3 c.c. of liver extract per day. No further increase is made. This is given for 20 days. Sometimes we get very good results with this line of treatment.

Another useful procedure is to give Testosterone Propionate 100 mgms. daily for 12 days. At the end of 12 injections, 12 more injections are given weekly twice. This hormone improves the appetite, fixes glucose in the liver to form the protein. It also produces a sense of well-being.

In the last seven years I have been using Eclipta Alba leaf extract in the treatment of cirrhosis liver. I used to have the leaf crushed and juice extracted. For each ounce of the leaf extract 1/3 ounce of glycerine is added as a preservative. Half-an ounce of this extract is given thrice daily before food. The same effect is achieved by using the dry leaf powder. At my suggestion, it has been made in a pill form from the dried leaf extract by Messrs. Pharm Products, under the trade name of 'Anticirhos'. The dose is one pill thrice daily before food. Once the patient is accustomed to it, 2 pills 3 times a day before food is given. The effect of this is to produce a mild laxative action. The leaf has been analysed and found to contain Beta-cystosterol. We have been trying to get Beta-cystosterol in a powder form for control of cirrhosis liver without success. The effect of the leaf extract on the blood is to help the albumin moiety to rise without touching the globulin moiety. As soon as the albumin content touches 3·5 gm. the ascites clears.

Another useful procedure is to give the patient 1 pint of 5% glucose intravenously with insulin 10 units subcutaneously to fix the glucose. Repeated blood transfusion is also of very great value. The main difficulty is to procure blood for transfusion. For the treatment of coma, the best drug that I have used is Chlor. Tetracycline 200 mgms. in 5% glucose solution, 1000 mgms. of Vitamin-C, and 50 mgms. of Vitamin-K 100 mg. of B-1, and 300 mgms. of Nicot inicacid. If the patient is free from diabetes, ACTH 25-30 units may be added to the drip with advantage. The cloudy state of mind can be cleared by giving the patient

Glutamic acid. Hepatic coma can be corrected by early administration of blood transfusion and avoiding drugs like barbitone and morphine and also by intake of high protein and ammonium chloride.

Since diet is an important feature in the treatment of cirrhosis liver, we have worked out a diet suitable for Indian patients, which is followed in my wards for the last two years.

The diet is as follows :—

Skimmed milk	...	1 pint
Bread	...	6 ozs.
Sugar	...	1 oz.
Boiled rice	...	6 ozs.
Curds	...	½ pint
Oranges	...	2
Boiled vegetables	...	2 ozs.
Liver fry	...	4 ozs.
Biscuits	...	2 ozs.

This diet can be modified for vegetarians by avoiding liver fry and eggs and increasing the quantity of skimmed milk to 3 pints. 3 pints of skimmed milk if administered straight-off to a man who is accustomed to light diet may cause diarrhoea. So the quantity of milk must only be increased gradually. The main principle in the diet is to give carbohydrate diet with enough of protein but low in fats.

In summary, the treatment is to prevent the preventible factors like mal-nutrition, syphilis, Kala-azar, amoebiasis, to eradicate de-vitalising diseases like ankylostomiasis and tuberculosis, and to give relief to the patient with ascites by doing paracentesis abdominis, and support the damaged liver with lipotrophic factors and by the use of Eclipta Alba leaf.

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Aberrant Renal Arteries and Renal Blood Supply

Dr. T. JANARDHANAN, M.B.B.S., M.SC.*
& Dr. T. JAYAVELU, M.B.B.S., D.Anth.†

Aberrant renal arteries are common anomalies met with in an ordinary dissection or surgical practice, the kidney being supplied by more than one artery and from more than one source. These are developmental defects which occur as prearranged segmental blood supply not only to the kidney, but to organs developmentally contiguous like the suprarenals and the gonads; hence the importance of aberrant renal arteries.

MATERIAL AND METHODS

Materials have been obtained from routine dissection hall specimens of 18 cadavers examined during one session at the dissection hall. Two specimens were found to project this altered morphology in the kidney. In addition to ordinary dissection of the specimens, radiograms have been taken to show the arterial arrangements inside the kidney, the contrast medium used being red lead injected into the vessels as for routine preparation of specimens for dissections.

DESCRIPTION

Specimen I.—Fig. 1. The left kidney is at a lower level than the right; it is flattened and irregular; sinus is defective. The anterior lip of hilum is not formed. The pelvis is small and divides on the surface of the kidney into a short lower and a long upper major calyx. The short lower calyx divides in turn into four minor calyces, which disappear from surface view; the upper divides into three only; these run a short distance before disappearing into the kidney substance. The usual arrangement of calyx, artery and vein at the hilum is disturbed.

*Professor of Anatomy, Madras Medical College, Madras.
†Asst. Professor of Anatomy, Madras Medical College, Madras.

Two arteries of small size supply the lower pole of the left kidney with their origins from the anterior surface of the aorta at its bifurcation and from the lateral side of the left common iliac artery near its source respectively. Both these arteries run behind the ureter. From the level of the inferior mesenteric artery an artery of medium size arises from the aorta, on its lateral side, and supplies the middle of the kidney in its lower half. The upper half of the kidney is supplied by another vessel that arises from the aorta laterally below the level of the superior mesenteric artery.

In the right kidney the hilum is better formed though not normal. The pelvis is poorly formed as in the left. However the division of major and minor calyces is well seen.

Two arteries supply the right kidney. The upper arises below the superior mesenteric artery from the lateral side of the aorta, runs behind the inferior vena cava and follows the upper major calyx. The lower artery arises from the anterior surface of the aorta runs behind the inferior vena cava and follows the lower major calyx. The lower artery in addition gives off the testicular supply. There is no other vascular supply to the testis on this side.

Specimen II.—Figs. 2 & 2(a). On the left side, the kidney and renal vessels conform to normal pattern.

On the right side the kidney is normal in appearance. It is supplied by two renal arteries arising from the lateral side of the aorta. One arises from the upper border of the 2nd lumbar vertebra, divides into two branches, one of which enters the anterior surface of the upper pole, the other passes through the hilum. The apical branch also supplies two phrenic twigs and a suprarenal. Another artery arises from the lower border of the 2nd lumbar vertebra, divides into two branches and enters the hilum clasping the main renal vein.

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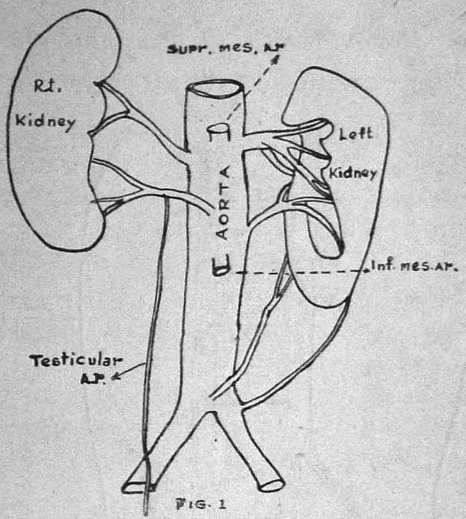
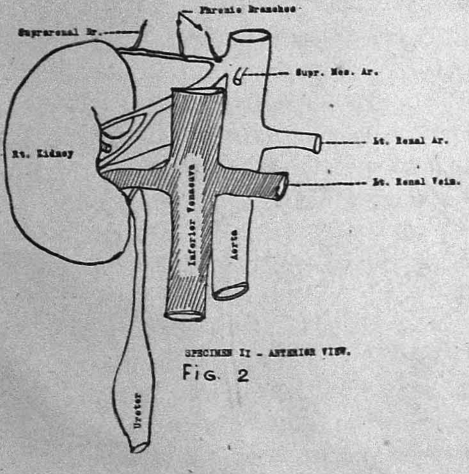


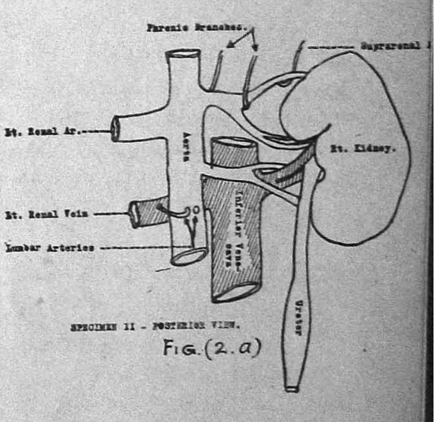
FIG. 1

SPECIMEN .I. ANTERIOR VIEW.



SPECIMEN II - ANTERIOR VIEW.

FIG. 2



SPECIMEN II - POSTERIOR VIEW.

FIG. (2. a)

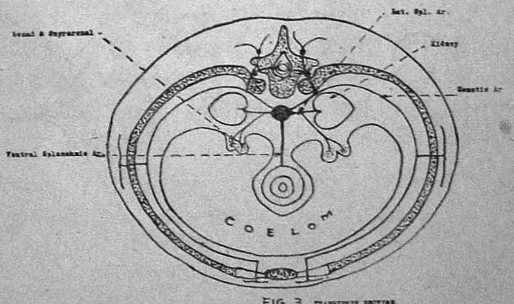


FIG. 3 TRANSVERSE SECTION.

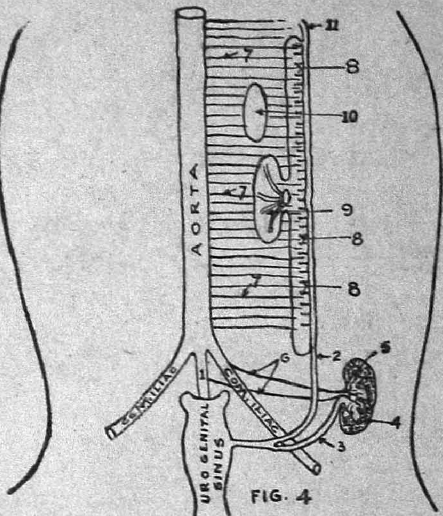


FIG. 4

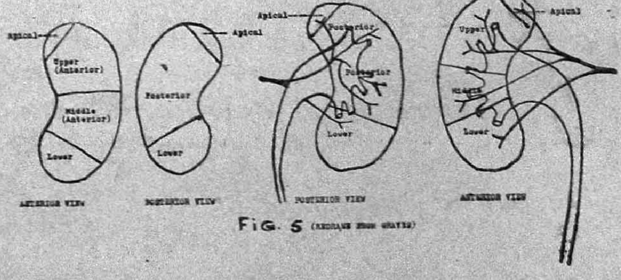


FIG. 5

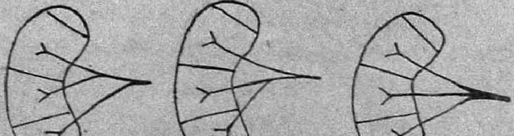


FIG 6

NORMAL ARTERIAL SUPPLY

The renal artery on either side arises a little below the superior mesenteric artery; the right arises a little lower than the left, runs horizontally at right angles to the aorta behind the renal vein, inferior vena cava, head of the pancreas and the duodenum. The left arises a little higher to right, lies behind the left renal vein, body of the pancreas, splenic vein and probably crossed by inferior mesenteric vein. As the arteries, both right and left, enter the hilum of the kidney, they divide into four or five branches. Most of the branches lie between the renal pelvis and renal vein. One or more branches run posterior to pelvis. Both give inferior suprarenal branches.

DEVELOPMENT

Figs. 3 & 4. The adult kidney is developmentally a finished product of two elements *viz.* nephrogenic cord which contributes to the non-segmental metanephric cap, the secretory part, and the diverticular part of mesonephric duct which forms the collecting tubules, calyces, pelvis and ureter, the conducting part. The adult renal arterial supply comes from the remnants of the mesonephric arteries secondarily mustered into for the function of renal supply. The kidney is originally pelvic with a plexiform network of vessels supplied by the common iliac and the pelvic part of aorta (median sacral artery). As the kidney ascends to its adult lumbar position this plexiform network of vessels transfers its dependence to the mesonephric arteries which are the lateral splanchnic vessels. The vessels in addition supply part of the future diaphragm, the gonads, suprarenals, sympathetics and lymphatics of that territory. A good many of mesonephric vessels do not degenerate with mesonephros for this reason. These arteries are not paired or segmental. In the adult they extend between the superior and inferior mesenteric vessels. This group of arteries could be classified into three sub-groups with reference to suprarenal which is lumbar in position both in the embryo and the adult. They are:

(*i*) those that run cephalad and dorsal to the suprarenal called cranial group.

(*ii*) those that pass through the substance of suprarenal called middle group ;

(*iii*) those that pass caudad and ventral to suprarenal called caudal group.

Finally each group is reduced to about two or three from the original thirty or more vessels that form the entire series. The groups form a mesh work of anastomosis between them. The first group gives rise to phrenic vessels ; the second group to suprarenal and the third group to gonadal and renal vessels. It is obvious from this that gonadal vessels would lie ventral to the kidney and the ureter. It is possible that the original plexiform network supplying the kidney may persist explaining the divergent source of renal supply. Occurrence of interdependent vessels to suprarenal, gonads and kidneys are explained from the above facts. Anterior lip of hilum is absent in horseshoe kidneys and kidneys with multiple renal arteries. Partially ascended kidneys often retain their embryonic median sacral and common iliac blood supplies.

Arterial Segmental Supply to Kidney.—Figs. 5, 6 & 6-(a). There are five segments of arterial supply to kidney, *viz.* apical, upper (anterior) middle (anterior), lower and posterior segments. This pattern is constant whether supplied by a single vessel or multiple vessels. There is no collateral circulation between them. Each segment is supplied by its own vessel. The anterior division of the main renal artery gives off branches to upper, middle and lower segments. The apical segment is usually supplied by the anterior division. The posterior division of the main renal artery supplies only the posterior segment. Occasionally it may give an accessory contribution to the apical segment. The posterior division does not show variation except for its apical contribution. The anterior vessel on the other hand shows variations in its division into segmental tributaries. These fall into three broad groups as shown in figure No. 6.

Aberrant Renal Arteries.—Strictly said the aberrant renal vessels can only come off from other sources than mentioned under development *e.g.* the mesenteric artery or intercostal,

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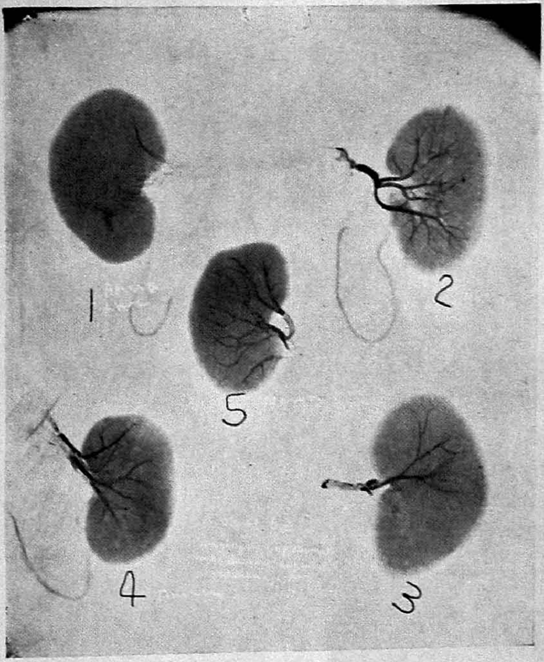

Fig. No. 6a.

Angiogram of Normal and aberrant Segmental Vascular Patterns

1. Apical and Lower Segments.
2. Posterior Segment.
3. Upper Anterior Segment.
4. Anamolous Segment.
5. Normal—All Segments.

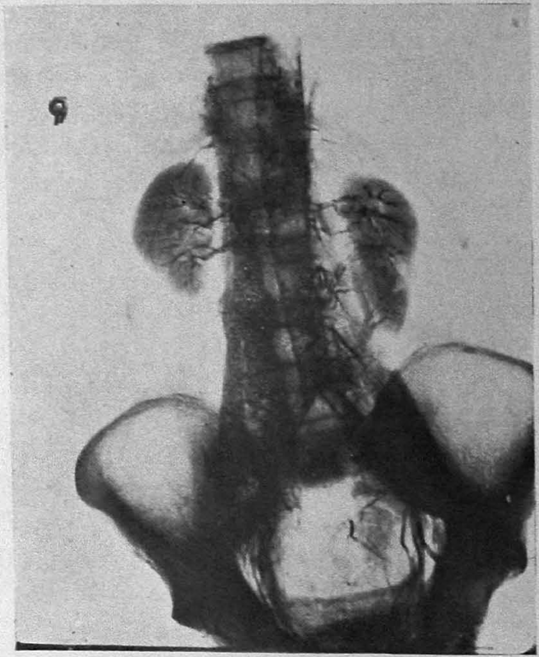

Fig. No. 7
Angiogram—Specimen I

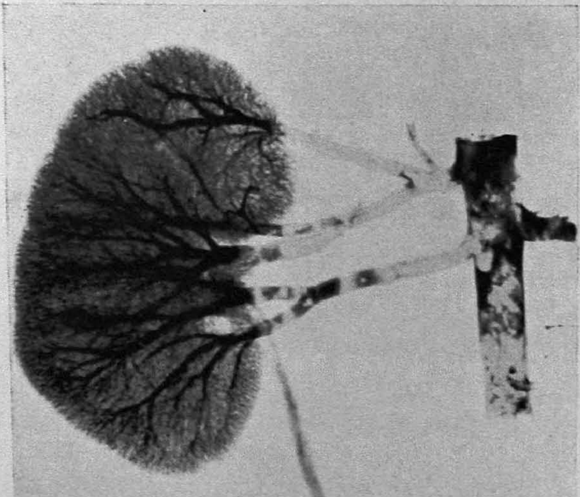

Fig. No. 8—Specimen II

lumbar etc. arteries. The occurrence of aberrant vessels in the kidney is about 31%[4]. The aberrant vessels are segmental vessels with a precocious origin. Arteries for the lower pole is more common; it is "twice the ratio for the upper pole". This is explained by the fact that the independence of embryonic vessels towards the caudal end of kidney is retained, whereas at the cephalic end they tend to coalesce. The artery for the upper pole always enters through the anterior surface, never the posterior. The lower polar arteries take an indifferent course. Supernumerary vessels may arise from phrenic, suprarenal, gonad, main renal or aorta. If it be from sources other than aorta it is common for the upper pole to be supplied separately. Independent renal vessels from aorta confine their extent between the origins of mesenteric vessels. Their course may be pre-caval or post-caval, close to it or away from it. The lower polar arteries may be a source of ureteric compression coursing as they usually do in front of the ureter.

DISCUSSION

Specimen I, Fig. 7.—As said above the lower pole arteries of the left kidney confirm the fact of their common occurrence. From their dorsal position to the ureter and from their origins, it is evident that the lower polar arteries of left kidney are its persistent embryonic pelvic blood supply. The accessory renal arteries on both sides are explained developmentally. The right testicular supply from one of the aberrant vessels can also be reasonably explained. The radiogram of this specimen confirms the segmental nature of the aberrant vessels, as given below:

Lt. Kidney.—The lower segment is supplied by the two vessels arising from the aorta and common iliac. The upper and apical segments are supplied by the upper vessel. The lower vessel supplies the lower and posterior segments. It is not clear how the middle segment is supplied as it is very poorly developed, perhaps by the upper vessel.

Rt. Kidney.—The upper artery supplies the apical, upper and the whole of the posterior segments. The lower artery is nutrient to the other two segments.

Specimen II, Fig. 8.—Here again the segmental division is evident from the angiogram. The upper artery supplies the apical, upper and posterior segments. The lower artery covers the other two territories.

The arteries to the kidney, suprarenal, testis and diaphragm inherit an embryonic interdependence. Even in the adult normal anatomy this fact is vividly manifest. The inferior suprarenal arteries come off from the renal ; the gonadal vessels near its origin give off small twigs of supply to the perirenal fat and the contiguous part of ureter. Even in the phrenic there is a divergence from the normal as it may arise from the renal. This is seen to be the fact in the first specimen. It is extremely important therefore that interdependence of these viscera for nutrition should be paid greater attention.

CONCLUSION

Aberrant renal arteries are sources of surgical hazard as they may involve the nutrition of other viscera like suprarenal or gonad. Because of their common occurrence, as one in three individuals, it will be rational as a routine to do an angiogram of the kidney before surgical interference for partial or total resection. Stone formation in the lower polar region of the kidney is more common and it is also this pole that is often supplied by an independent aberrant blood vessel. This vessel may sometimes be a common source of supply to kidneys and gonads, increasing the risk of lower polar resection of kidney. Therefore an angiogram will not only help to predetermine the segmental supply of aberrant vessels, if present, but will also exclude the imminent danger associated with renal vascular alterations. A surgeon may well be cautioned also about the persistence of embryonic longitudinal venous channels like double inferior vena cava or the left inferior vena cava.

From the anatomical aspect it will be of interest to mention that developmentally there are about 20,000 lobules in the kidney forming into eight to eighteen pyramids derived from about 20 generations of ureteric division. The pyramids open

into about eight minor calyces and they into two or three major calyces and finally into the ureter. Such extremely complex division yet begins only in the unsegmental metanephric cap and Wolffian diverticulum. What will be the relation between foetal lobulations of the kidney and ureteric division? What position the foetal lobes finally come to take in the kidney? How the adult arterial pattern will compare with foetal lobular arterial pattern? Answers to these will be definitely instructive.

SUMMARY

Two cases of aberrant renal arteries, in one of which there is a testicular supply from the renal, are discussed.

Normal segmental arterial supply, embryology and segmental nature of the aberrant vessels are indicated.

The necessity for a routine renal angiogram in cases of kidney resection is emphasised.

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Syphilis and Pregnancy

Dr. P. N. RANGIAH, M.D.*

INTRODUCTION

Mr. President, Ladies and Gentlemen,

I must thank the kind sponsors of my name to the Syndicate of the University for inviting me to deliver the series of Dr. Elizabeth Mathai Memorial Endowment Lectures. I have never felt so embarrassed as just now. I sometimes wonder if there is anything like a 'solo' presentation of credit to place before you by me. However, with blessings of my Chief, Dr. R. V. Rajam, the guiding star of the Institute of Venereology for the last three decades and good wishes of the large bevy of medical officers, named seriatim—Dr. (Miss) P. Samuel, Dr. (Mrs.) F. Mathuranayagam, Dr. (Miss) Noronha (now Mrs. Masilamani), the late Dr. (Mrs.) Selvanayagam, Dr. (Mrs.) Abraham, Dr. (Mrs.) Anguli, Dr. (Miss) F. Iswariah, Dr. (Mrs.) Ramachandra, Dr. (Miss) C. N. Sowmini and Dr. (Miss) Vijayalakshmi—who all as a team have worked assiduously in the management of the patients and the maintenance of records, I am confident I shall have more than enough of 'the joint property' for exhibition before this learned audience. The first topic I would like to touch upon is entitled "Syphilis and Pregnancy". I have no doubt in my mind that the terms "Syphilis and Pregnancy" are familiar to everyone of the audience gathered here.

SYPHILIS IN SPECULATIVE PHASE

It may be recalled at this time that syphilis is said to have appeared on the horizon of human mind when Columbus returned to Europe with his sailors from America in 1494.

*Associate Professor of Venereology, Madras Medical College, Madras-3.

Thereafter for nearly 400 years much about syphilis was speculative in character. The cause of the disease was little known. The diagnosis depended very much on clinical acumen and judgment based on the morphological characteristics of the very many manifestations of syphilis.

A FEW STRANGE LAWS

No wonder certain strange laws like Colle's law and Profeta's were enunciated by the men of the day. To-day, it would appear very fantastic that a congenital syphilitic baby should be taken as conferring immunity on the mother; or the syphilitic woman as truly protecting her new born from syphilitic infection; for the serological tests for syphilis have shattered the misconceptions by discovering the presence of syphilis in the apparently normal mother or the well looking baby. "No syphilitic mother, no syphilitic baby" is an aphorism true even to-day.

THE CONCEPT OF THIRD GENERATION SYPHILIS

Yet another puzzling hypothesis current in the speculative era was that syphilis was passed on to the third generation. Several case histories are on record in the literature pertinent to and suggestive of support to this hypothesis of third generation syphilis. Even this concept seems very much to have been based on clinical suspicion. Over a period of a quarter of a century of association with the Institute of Venereology, it has not been the experience or the good fortune of the staff to encounter even a single case of incontrovertible third generation syphilis worthy of record.

SCIENTIFIC PHASE OF SYPHILIS

The change over from the speculative era to the scientific phase of syphilis dates back to the year 1905 when Schaudinn and Hoffmann demonstrated the etiologic agent of syphilis and gave it the name, Spirochoeta pallida or Treponema Pallidum. Within a year of demonstration of the causative agent, Wassermann working on the lines of Neisser and Bruck's experiments,

introduced the complement fixation test for the diagnosis of syphilis. Just a little earlier, to be certain in 1895, Roentgen had already discovered for the world the x-ray ; this has added to the knowledge of the syphilologists, through placing at their disposal the means to visualise the wrecked state of the skeleton in syphilis hitherto concealed. In the field of therapy, Ehrlich's discovery of salvarsan or what was popularly known as 606 in 1912 as " the one injection cure " or the therapia magna sterilizans for syphilis, happened to have held out the hope of speedy and reasonable cure to the syphilis-stricken population. It is clear to us thus that in the scientific era the diagnosis and the treatment of syphilis came to be placed on a satisfactory plane ; such that very soon it became apparent that the once dreaded 'big pox', syphilis, was under siege.

DECLINE OF SYPHILIS WITH DISCOVERIES

That the conquest of syphilis had begun was the consensus in the year 1936 when Jadassohn (Medical Clinics, Vol. 33, 1936) published that 51 eminent specialists of 19 countries tabled a statement that syphilis was on the decline, based on figures obtainable in those days ; the decrease in the incidence of syphilis in England was 50%, that in France was about the same. A review of literature since that period brought up-to-date, is replete with the monotonous repetition of similar expressions of a fall in syphilis rates, both in England and in America. To conclude, syphilis for long has been pronounced a dying disease! Yet it has survived with periodic upsurges! Ehrlich's salvarsan or the successive arsenical refinements really failed to effectively pin down syphilis.

SYPHILIS, THE INFANT KILLER

Syphilis has been the enemy of mankind particularly of the unborn offspring of pregnant women. Our country is credited with a very high death rate, one of the causes for the intra uterine foetal or early infantile mortality being attributed to untreated syphilis in the pregnant women.

Here I may pause to remark that while our country is seized with the problem of the phenomenal rise in population figures and is all out in the attempt to plan parenthood and make the business of pregnancy a limited concern, it may seem paradoxical that the subject for discussion should have a bearing on the control of the death rate by checking one of the etiologic factors, syphilis—notorious as you will admit, for its fatal-influence on pregnancies and neo-natal phase of infancy. Alone, syphilis or pregnancy may not interest anybody so much as when both overtake a woman simultaneously.

STRANGE FEATURES OF DUAL INVASION OF THE OVUM

A man infected with and in the communicable phase of syphilis is likely to transmit both the treponema pallidum, the causative agent of syphilis and the spermatozoan, the necessary component for conception, into a healthy, uninfected woman with the result that the woman partner in sex partnership may not only fall victim to syphilis but also become pregnant concurrrently. The curious thing, however, is that the third party, springing out.of the partnership, the developing foetus, is not infected all at once. While the treponeme takes the seemingly high road and the sperm takes the low road, both do not meet at the end of the road—the ovum—for a long time!

Pathologists declare from their observations that the treponema pallida deposited in the genital tract of the women, take to the blood stream, primarily or secondarily *via* the lymph, and are emphatic that the treponemes in the maternal circulation are incapable of reacting on the developing zygote, immediately. Full 16 weeks of pregnancy, they presume, must elapse before a transplacental transmission of the syphilitic infection to the foetus can occur. Reasons given are that the foetal circulation is not well developed till 16 weeks of conception and that it is just at this time of pregnancy the cells of cyto and syncytial trophoblast get thinned out considerably to enable the treponemes to traverse the placenta.

To the thinking syphilologists, these arguments seem strange! Why should treponema pallidum noted for its capacity to penetrate intact skin or mucous membrane be reckoned incapable of effecting entry into the placenta till the 16th week of development of the foetus? However, the entry of the spirochaetes into the foetus must occur earlier than the time of appearance of histopathological changes, pathognomonic of syphilitic infection in the placenta.

Histopathologic studies in syphilis of the foetuses at various age periods of foetal development, judged by the measurements of the foetuses and their weights, do not reveal evidence of syphilitic infection in the sections of foetuses, corresponding in age to periods of less than 16 weeks, nor has the treponema pallidum been seen in these studies. To digress, admittedly, therefore, it is the consensus that the death of the foetuses earlier than the 16th week of development is not etiologically related to syphilis.

OBSTETRICAL DISASTER

The influence of syphilis on the developing zygote after the 16th week.—After the 16th week of development of the foetus, any of the many unfortunate syphilitic episodes may occur. The obstetrician describes them in such terms as 'premature, live born' or if 'dead born', he categorises them as 'miscarriages', and 'still births' and if born alive to later die within a month of the birth, he classifies the outcome of pregnancy as "neo-natal death".

An exception to the rule is that there may be an occasional escapade from the routine list of disasters such that an infant 'born alive at full term', 'natural delivery' may not exhibit any evidence of syphilis at any time; or the baby born may not manifest signs and symptoms of syphilis till late in childhood, adolescence or adulthood when the syndrome is classified by the syphilologist as tardive heredo-syphilis.

The observations independently made in the reverse direction through autopsy studies confirm the deplorable influence of syphilis in pregnancy:

CHART I

Autopsy Studies

Particulars	Total examined.	Total syphilis.
1. Rathnavathy and Reddy ...	164	3%
2. Arey and Dent (non-macerated foetuses and 102 live born infants dying in the neonatal period) ...	35	9%
3. Arey (alone) study of prematurely born ...	22	5%

INFLUENCE OF PREGNANCY OVER SYPHILIS

It is claimed paradoxically that pregnancy exerts a beneficial influence on syphilis. The clinical manifestations of syphilis in a pregnant woman are noticed to be mild or that they may be absent altogether. Multiparity in a syphilitic woman may reduce the chances of the woman's developing late manifestations of syphilis. Moore found many positive spinal fluids in women with late syphilis, who had not been pregnant after the infection ; this was not the finding in syphilitic women who had gone through pregnancies.

Solomon recorded the high incidence of sterility in neuro-syphilitic women ; it is presumed these women neuro-syphilitics lacked the beneficial effect of pregnancy.

Again, the observation is recorded that after treatment of syphilis in women in the arsenical era clinical progression in multiparae occurred to the extent of 4·1% but 7·1% in nulliparae. Women treated for latent syphilis showed progress or relapse to the extent of 0·7% in the pregnant group and 5·4% in the non-pregnant group.

An identical experience is recorded in a study of experimental syphilis. Two batches of rats, 8 each per batch were put to an experimental inoculation of spirochoetal emulsions. This resulted in the production of syphilomas in 3 to 4 weeks, amongst the non-pregnant group. In the pregnant group, on the contrary, no lesions developed in 4 of them. An infiltration occurred at

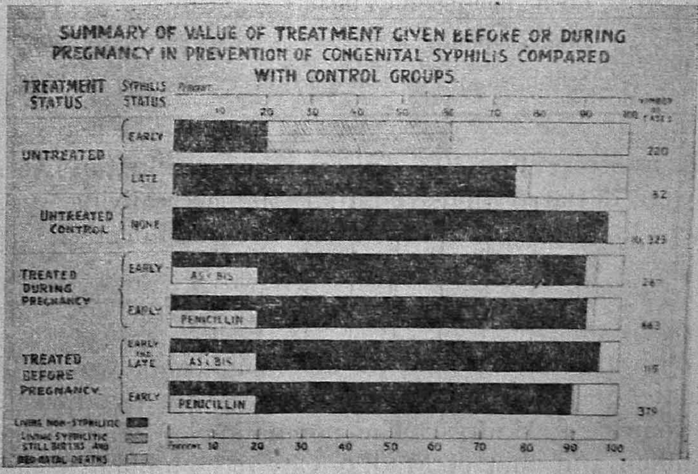
the site of inoculation in 3. In only one, a lesion corresponding to the type in the control group developed during the period of lactation.

Certainly these findings lead to the conclusion that pregnancy exerts an apparently moderator influence in syphilis judged by the external manifestations of the disease in the experimental animals.

Jonathan Hutchinson was the first to record such an observation in his clinical work. It is not a wonder then that students of syphilis have been tempted to prescribe pregnancy for syphilis in women. Pregnancy probably alters the immunity reaction leading to the production of insignificant lesions or allows the disease to sink beneath the horizon of clinical recognition. Our opinion is not in conformity *vide* clinical photo.

A reference is necessary to the Chart II depicting the outcome of pregnancy in syphilis as per studies of Ingraham of Philadelphia with an 'explanatory note on statistics on the outcome of pregnancy in syphilis':—

CHART II



In a control group of pregnant women in whom the possibility of syphilis has been excluded, it has been pointed out that healthy full term live births occurred in 86%, the remaining 14% being variously allotted under terms of still birth, prematurity and neo-natal deaths.

The results of untreated early syphilis in pregnancy are notoriously bad. Live born non-syphilitics are just 18%. The remaining 82% comprise syphilitic babies 40% and the residuum composed of prematurity, still births and neo-natal deaths.

What are our figures? No large scale experimental study of this sort has been carried out in our area. Sometime ago a statistical analysis of the outcome of a short series of cases of 274 pregnant women with syphilis was carried out.

CHART III

Our experience

Analysis of outcome of pregnancy in 274 untreated syphilitic pregnant women

	Number of pregnancies	...	274
Outcome :	Living non-syphilitic infants	...	40%
	Living syphilitic infants	...	30%
	Still birth and neo-natal death	...	30%

It transpires that 40% of pregnancies resulted in live births and were declared non-syphilitic, 30% were living syphilitic infants and 30% were still births and neo-natal deaths.

SYPHILIS IN PREGNANCY

With this background at this juncture I would like to speculate in mathematics and pose a few questions about the women of Madras. The population of Madras State is 35 million and that of Madras City is 1·4 million. Calculated

by the ratio given in Dr. Chandrasekar's book, the number of females in Madras City alone works out at 6,78,900. How many of these women are in the reproductive period of age and how many births are expected out of them per year?

CHART IV

Population figures in Madras for 1951

Madras State ...	35,734,489
Madras City ...	1,416,056
Number of females in Madras City ...	678,900
Number of births ,, ...	74,000 (anticipated)

The statistician of the Institute of Venereology applying certain formulae opined that there could be 74,000 pregnancies in a year, which is close to the figure recorded in the Annual Report of the Health Officer of the Corporation of Madras. How many among these pregnant women are syphilitic? To know this, one has to pore through the pertinent statistics available.

It turns out that ordinarily 5·7% of pregnant women are syphilitic, judged by the figures furnished by the Lady Pathologist, Corporation of Madras, working in close collaboration with the Lady Superintendent of the Maternity and Child Welfare Centres functioning under the supervision of the Health Officer of the Corporation of Madras.

CHART V

Percentage of Positive Reactors to Syphilis

Madras Corporation Antenatal Clinics

YEAR	Total No.	Percentage
1953 ...	8692	6·4
1954 ...	13393	6·6
1955 ...	15179	6·1
1956 (6 months) ...	6588	5·51
1957 ...	13425	5·1
1958 (6 months) ...	9256	4·6
Total ...	66533	34·31/6 *i.e.* Average 5·7%

The information furnished by the Lady Superintendent of the Government Hospital for Women and Children, Egmore, Madras, reveals that the positive reactors amongst the pregnant women during the antenatal clinics constitute 8·3%.

CHART VI

Percentage of Positive Reactors to Syphilis

Government Hospital for Women and Children, Madras

YEAR	Total No.	Percentage
1953 ...	11736	6·3
1954 ...	8824	8·6 (March to Dec.)
1955 ...	10276	8·58
1956 ...	9561	8·4
1957 ...	6162	9·47
1958 (6 months) ...	5136	8·58
Total ...	51695	49·03/6 i.e. Average 8·3%

A third source of information is the Madurai Municipality that reported the percentage positivity amongst the pregnant women attending the clinic as 13%.

CHART VII

Percentage of Positive Reactors to Syphilis

Madurai Municipal V. D. Clinic—Antenatal cases

YEAR	Total No.	Percentage
1955 ...	6340	11·9
1956 ...	8374	13·5
1957 (10 months) ...	8774	13·8
Total ...	23488	39·2/3 i.e. Average 13%

If the pregnancy rate among the women of Madras City is what it is as per Dr. Chandrasekar's disclosures and if the proportion of syphilitic pregnant women as per samples attending the different ante-natal clinics be high by calculation, we have to anticipate a stupendous quota of syphilitic pregnant women in our city and much more for the State every year.

The final question now posed is, do all the syphilitic pregnant women come under the syphilologists' purview for treatment? No. Most of them go elsewhere to be seen by non-specialists whose index of suspicion for syphilis is regrettably low. Only if history suggests syphilis STS is done ; when women go in with labour pains STS is not done at all and syphilis is forgotten once for all, as soon as the pregnancy is over.

Here lies the risk. Syphilis is likely to be missed in a large majority of the pregnant women, for nearly 40%, as per the statistics of the Institute of Venereology (*vide* Chart VIII), do not carry any arresting signs of syphilis on their exterior. These women are classed as latent syphilitics and can be discovered only by serological tests for syphilis performed on them. It is imperative on the part of every member of the medical profession attending on a pregnancy case that a serological test for syphilis shall be insisted on. A positive serological test for syphilis may for practical purposes be taken to mean syphilis in the pregnant women.

In passing, an explanation is necessary for the variation in percentage positivity furnished by the three different groups of ante-natal clinics. The number of positive reactors amongs pregnant women attending the Maternity Hospital is on the increase every year while there is a decline in the number of positive reactors attending the Corporation Maternity centres. Lack of facilities for treatment of syphilis and follow-up of treated patients may explain the incongruity.

ANALYSIS OF THE OUTCOME OF PREGNANCY IN TREATED SYPHILITIC PREGNANT WOMEN

A further reference to the figures furnished in the chart by Ingraham of Philadelphia, relative to the outcome of pregnancy in syphilis treated with penicillin reveals results far superior to those observed in normal non-syphilitic pregnancies.

Our observations limited to small groups of pregnant women with syphilis are similar and are recorded as under with remarks on the interesting high lights.

REMARKS ON THE TREATED SERIES AT THE INSTITUTE OF VENEREOLOGY

MARITAL STATUS

It is curious that there were only 4 prostitutes in the list. Probably it is difficult to question and determine the status of the women concerned.

Kept mistresses equivalent to unmarried women numbered 149.

Married women constituted the biggest bulk—677 of them.

STAGE OF INFECTION

There were very few cases of primary syphilis detected among pregnant women. The paucity may be explained by a hypercritical syphilologist as a pseudo rarity resulting from a reluctance on the part of the diagnostician to examine carefully, coupled generally with (1) the difficulty in the examination of women and (2) the not easy accessibility of the genital lesions in the women.

A large majority of the pregnant women had secondary syphilis—460 of them amongst 846.

Tertiary syphilis was less frequently encountered.

The remaining pregnant women were in a state of latency.

CHART VIII

Correlation of Different stages of Syphilis in Husbands of 307 Pregnant women having different stages of Syphilis

Institute of Venereology 1948—1957

Status of the Wife	Status of the Husband					
	Sy. I	Sy. II	L. S.	Sy. III	Seronegative	Total
Sy. I ...	7	9	12	—	7	35
Sy. II ...	25	44	47	—	25	141
Sy. III ...	1	2	3	—	1	7
Latent Syphilis ...	14	24	47	2	37	124
Total ...	47	79	109	2	70	307

The clinical and serologic status of the partners have been incongruous, seeing that there were 70 non-reactive husbands when their consorts reported with manifest syphilis; it may be that the so called wives may have contracted syphilis outside the marital fold or that the husbands after communicating the infection to their respective wives took treatment and turned non-reactive subsequently.

CHART IX

Age distribution of 846 pregnant women who attended the Institute of Venereology during 10 year period 1948-1957

AGE GROUP	Number
Below 15 years ...	33
16 to 20 years ...	365
21 to 25 years ...	263
26 to 30 years ...	139
31 and above ...	46
Total ...	846

The age of the large majority of the pregnant women with syphilis ranged from 16 to 30. There were 33 women below 15 years.

CHART X

Number of cases Infected during the particular pregnancy

Year	Total Cases	1st para	2nd para	3rd para	4th para	5th para	6th para	7th para	8th para	9th para	10th para
1957	65	17	12	14	5	6	7	1	2	—	1
1956	54	15	6	12	6	7	3	5	—	—	—
1955	83	22	24	9	13	7	3	1	1	—	3
1954	118	30	30	26	13	7	7	2	1	—	2
1953	106	33	23	22	7	8	3	8	2	—	—
1952	102	30	27	22	8	7	4	—	2	1	1
1951	131	33	23	19	15	4	6	6	4	1	—
1950	57	17	23	6	7	—	2	1	—	1	—
1949	60	21	16	5	8	6	2	1	—	—	1
1948	70	22	17	12	7	5	2	1	—	3	1
10 years.	846	260	201	147	89	57	39	26	12	6	9
% ...	100	30·7	23·8	17·4	10·5	6·7	4·6	3·1	1·4	0·7	1·1

With reference to syphilis and parity, it is seen that syphilis occurred even in the 10th pregnancy. A woman patient with congenital syphilitic baby in her arms was exhibited to the audience and that was her 10th born baby.

The large majority of syphilitic pregnant women appear to belong to the first and second para. It is surmised that this is so because syphilis is overlooked in the would be partners in marriage in our country as they do not have to produce the pre-marital " freedom-from venereal diseases " certificate.

It may also be due to the convention that the newly wedded bride leaves her husband's house for her mother's in the later half of her first pregnancy ; and also goes out to her mother's place for the second confinement too. These periods of absence of the wife are considered to be most dangerous phases in the life of the bridegroom ; for it has been our experience borne out by the historical records of our patients that the grooms get sexually imprudent and stray away at such

times. Acquiring syphilis extra maritally thus, they freely award their home gone and returning wives, syphilis as a reinforced wedding gift.

Yet another circumstance predisposing to the acquisition of syphilis by the bridegroom and influencing the bride's early pregnancies is the vexatious custom that during the Tamil month of Adi, the bridal couple are restrained and refrain from sexual congress. This taboo offers temptations and the bridegroom oversteps the marks of prudence, succumbs to temptations slyly or otherwise, elsewhere, and passes on the acquired syphilis to the innocent bride and through her to the unborn foetus in the womb. Can we suggest a change in the customs and conventions of our people to halt this evil? This is essentially a sociological problem requiring attention.

CHART XI

Number of Cases Infected during each Trimester

YEAR	Total No. of Cases	First three months	Second three months	Third three months
1957 ...	65	8	35	22
1956 ...	54	12	24	18
1955 ...	83	15	42	26
1954 ...	118	10	65	43
1953 ...	106	14	55	37
1952 ...	102	16	41	45
1951 ...	131	32	75	24
1950 ...	57	7	33	17
1949 ...	60	6	29	25
1948 ...	70	4	40	26
Total ...	846	124	439	283

Our observation as from the above table indicates that most pregnant women have been diagnosed as having syphilis in the second trimester; only a few in the first trimester. There were 283 women diagnosed as having syphilis in the third trimester of pregnancy. This is not all. Syphilis was diagnosed in the pregnant women just about the time of delivery or soon after the termination of pregnancy in a number of cases.

CHART XII

Serological titre of the latent syphilitic pregnant women who attended the Institute of Venereology during 1953—1957 (Five Years)

TITRE				NUMBER
VDRL Pos.	1 Dils	...	22	(57) below 4 dilutions and 4 *i.e.*, 40·4%.
"	2 "	...	15	
"	4 "	...	20	
"	8 "	...	29	(84) 8 dilutions and above *i.e.*, 59·6%.
"	16 "	...	27	
"	32 "	...	16	
"	64 "	...	10	
"	128 "	...	2	
	Total	...	141	

The above table gives the serologic status in pregnant women with syphilis. 40% of them had low titre while the rest had serologic titre indicating 8 dilutions or above. It can be imagined how if the sensitivity of the serologic test had been poor, the 40% positive reactors in low dilutions would have been missed.

CHART XIII

Outcome of Treatment with Narrow Spectrum Antibiotic

PARTICULARS		Year 1953 to 1956
Total no. of pregnant women Adequately treated: 4·8 to 6·0 m.u. of PAM. or 2·4 m.u. of Benzyl amine penicillin	...	395
Outcome available in 179 cases:		
Living normal healthy	...	174
Living syphilitics	...	5

There is epidemiological evidence to suggest that 4 out of the 5 prenatal syphilitics born relate to re-infection in the mother. Hence there is only 1 failure.

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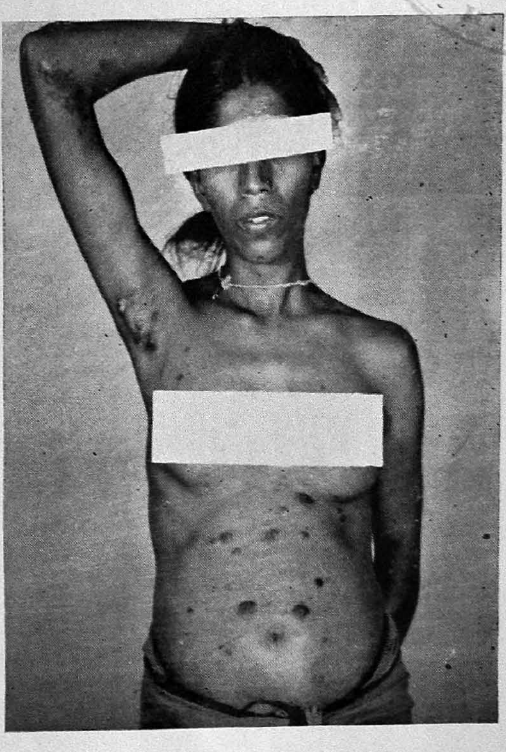

PROMINENT SYPHILODERMATA
in
a pregnant woman.

The above table records our experience with reference to the outcome of treatment of pregnancies in syphilitic women, between 1953 and 1956 with the narrow spectrum antibiotic. A total dose of 6 mega units of PAM in oil intramuscularly in divided doses of 600,000 units per day for 10 days was the standard schedule of therapy given. A small series of cases was each treated with 2·4 mega units of benzyl-amine penicillin in one single dose therapy.

Of 395 cases treated, 179 were available for assessment. All the 179 of them went through confinement, delivering healthy non-syphilitic babies except 5. The treatment failures in 4 out of the 5 pregnancies under reference were traced to reinfection of the pregnant women in the later half of their pregnancies, by their consorts.

Our experience during the years 1955–57 with the broad spectrum antibiotics, Achromycin, to be specific, is recorded as under:

CHART XIV

Outcome of treatment with broad spectrum antibiotics

PARTICULARS	Year 1955 to 1957
Achromycin Treated:	
Total No. of pregnant women ...	12
Clinical status—Secondary syphilis, besides D.F. was positive and high titre serology recorded.	
Outcome:	
Full term natural delivery ...	11
Foetal death due to breech presentation ...	1

The infants were followed upto more than 12 months and found to be clinically and serologically normal. Mothers followed up showed reversal of serology to negative in record time. Toxicity nil.

Achromycin (Lederle) for syphilitic women in pregnancy has been beneficial. Frank secondary syphilitic cases with dark-field positive lesions and high titre positive serology were

chosen for the purpose. Each of the patients was given 500 mgm. or 2 capsules of the tetracycline round the clock for 4 doses daily for 12 days. The cases were followed up and the record has been made that every one of the pregnant women delivered at full term, live, normal healthy babies. The babies were followed up, some of them for more than 12 months and declared free from syphilis. The mothers too were free from the infection and serologically reverted to normal, in record time of about 12 months. The only single mishap to a pregnant woman who ended her pregnancy in still birth was traced to a difficult breech delivery.

Syphilis in pregnancy is preventable. If not prevented it brings discredit to modern medicine.

CONTROL MEASURES AGAINST SYPHILIS IN PREGNANCY

Measures recommended for the control of syphilis in pregnancy :

1. Pre-marital S.T.S. should be enjoined when practicable.

2. Every pregnant woman shall be serologically screened twice in the course of her pregnancy, once in the early phase and another time in the late phase of her pregnancy. If seen in labour pains STS may be done soon after the delivery is over.

3. The quantitative serological test for syphilis is very helpful and should be insisted on in every positive case as it registers a rise or fall in titre in parallel with the progress or regress of syphilis.

4. Every positive reactor should become a focus of enquiry for the detection of syphilis in the sexual partners, siblings and other members of society and treatment when and where indicated should be given.

5. PAM in oil 2 c.c.—6 lakh units should be given intramuscularly daily for 10 days to every pregnant woman with syphilis. Epidemiologic dose of PAM should be delivered to contacts with no evidence of syphilis and standard treatment if found infected.

(**Note**.—In the arsenical era a syphilitic woman once given antisyphilitic treatment was retreated every time she became pregnant, throughout her reproductive phase of life. Such a necessity does not arise with penicillin therapy).

6. A pregnant woman treated for syphilis showing the slightest suspicion of reinfection requires re-treatment.

7. No period of time is too late for treatment of syphilis. Treatment is valuable even if it be started when the patient is seen in labour and identified as syphilitic.

Note.—We have not encountered any serious toxic reactions in pregnant women due to penicillin administration. Nevertheless we ought to be on the alert with the emergency kit. In cases of penicillin reaction, an injection of 1 c.c. of adrenaline 1 in 1,000 should be immediately given. Antihistaminics and corticosteroids to counter the reaction and supportive treatment to the heart and lungs are indicated and further observation be made.

8. Timely and adequate treatment of syphilis in a pregnant woman is the prophylaxis *par excellence* for congenital syphilis.

SUMMARY

A retrospective study was attempted on the subject of syphilis and pregnancy emphasising the influence of the one upon the other and *vice versa*, based on the experiences recorded hitherto in literature and at the Institute of Venereology.

That untreated syphilis has a deadly effect on the developing foetus is recorded. The remarkable effects of therapy of syphilis in the pregnant women instituted at the proper time with narrow or broad spectrum antibiotics, are well established.

It is emphasized that the birth of a congenital syphilitic baby tantamounts to a stricture on the Public Health Service under whose aegis the birth of a congenital syphilitic baby has occurred.

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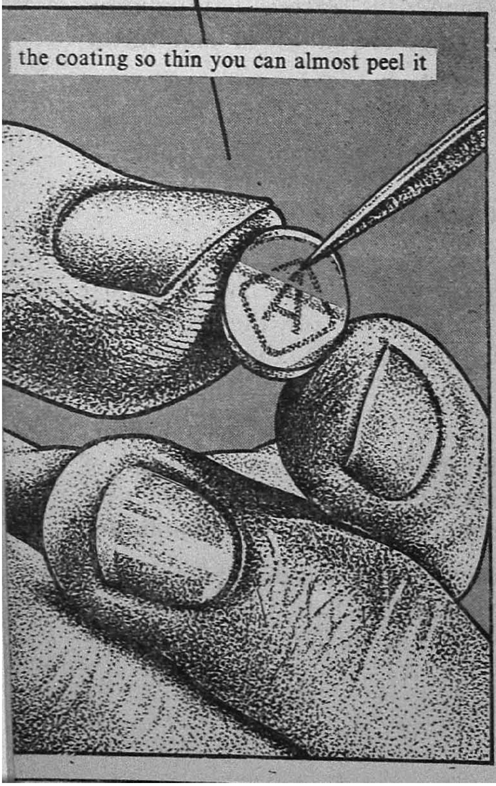

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Angio Cardiography

Dr. ARTHUR DANIEL, M.R.C.P.

Recent advances in the field of cardiac surgery, particularly in those congenital abnormalities of the heart, amenable to cardiac surgery have created a powerful stimulus for correct diagnosis of the developmental defects of the heart. In most of these, the diagnosis is made from careful physical examination along with simple roentgen examinations (screening and skiagram) correlating with electro cardiographic tracings. But accurate diagnosis of the number and nature of defects calls for cardiac catheterisation with pressure readings, blood gas, analysis and contrast visualisation of the chambers of the heart and blood vessels by angiocardiography. The use of the catheter has made selective angiocardiography a possibility along with cardiac catheterisation studies. This great advance in the knowledge of these abnormalities of the heart, is due to the work of Maude Abbot on autopsy specimens, the clinical studies of Brown in England and Helen Taussig in America, the angiocardiographic studies by Castellano et al and Robb and Steinberg and the surgical skill of Blalock, Logan and others.

The specialised investigation of angiocardiography may fail to interpret without the aid of other laboratory findings like the conventional x-rays, the electro-cardiogram and catheterisation studies. The dangers of this investigation are of such a nature demand to a careful selection of cases and proper preparations. The selection of cases is through a group discussion by the paediatrician, cardiologist, surgeon, aneasthetist and radiologist taking into consideration the risk of surgery and prognosis. Failure

to comply with the above may result in repeated investigations endangering the patient to radiation hazards. Further the positioning plays a vital role in bi-plane angiocardiography and this is done after careful investigations and detailed discussion. Needless to say that it is superfluous to subject to such investigation, evident and frank cases like atrial septal defect, pulmonary stenosis, and patent ductus arteriosus as these need proper positioning and timings without which the results may be misinterpreted. Gone are the days of mechanical operations; for triggering of these exposures at proper timings is done by a push button system with simultaneous electro cardiographic tracings and pressure injections.

The configurations of the cardiac chambers and blood vessels are visualised by the contrast medium. This anatomical consideration will reveal the presence and patency of chambers and vessels, size of these with their position and relationship to one another and abnormal communications if any. This is derived by the sequence of opacification, duration and intensity of opacification. So, the necessity for correct timings is apparent for considering the anatomical defects.

Having mentioned the one single indication for angiocardiography as careful selection of a case after group discussion, it is but proper to consider the contra-indications.

These are :—

1. Failure to meet the above requirements.
2. Hypersensitiveness to iodine.
3. Acute or severe chronic renal disease.
4. Allergy.
5. Acute febrile episode.
6. Thyrotoxicosis.
7. Dehydration, as this is worsened with the injection of the hypertonic dye.
8. Congestive failure.
9. Mongolism.

There are a few essential requirements or preparations necessary for proper angio-cardiography. The circulation time is useful though it is not absolutely necessary in rapid film method. The arm to lung time is arbitrarily taken as 4—8 seconds and for right heart circulation 2 seconds are deducted from this. Similarly, the arm to tongue time is 10—16 seconds and 2 seconds less will give the left heart circulation.

The timings are :

FOR INFANTS

Superior vena cava and right auricle	...	¼ – 1 second
Right ventricle	...	½ – 1½ seconds
Pulmonary artery	...	1½ – 2½ seconds
Pulmonary vein and left auricle	...	2½ – 3½ seconds
Left ventricle	...	3 – 4½ seconds
Aorta	...	3 – 5 seconds

FOR ADULTS

The timings are ½ – 1½ seconds more.

For aorta it takes 5—8 seconds.

The premedications used are hyoscine hydrobromide ½ an hour before, for adults 100 mg. of seconal or nembutol. Pethidine may be used. Morphia is not contra indicated, but since it is a respiratory depressant it is not advisable as the cause of death in some of these is by respiratory arrest. Anti-histaminics can be used in preparing the case. Anaesthesia is required for children under 10 years and not otherwise unless the patient is extremely nervous and non co-operative.

The sensitivity test for-iodine may be done in the ward but the patient may be put on pot. iodide mixture with the hope that it will prevent the allergic manifestations. It should be the routine method to inject 0·5 c.c. to 1 c.c. of the dye intravenously at the time of investigation and wait for 5 minutes to see if any allergic manifestations appear. But all these are only from consideration of the medicolegal aspect of the case.

The site of injection :

1. Left cubital vein for it shows the left superior vena cava which is otherwise missed.

2. Malleolar or saphenous vein in cases of vascular anomalies. The left anterior oblique position is chosen to avoid masking of these anomalies by the contrasted superior vena cava and also in atrial septal defects.

The open method is preferred in all these sites. Saline or citrate may be injected for patency of the canula and vein. The one single precaution called for, immediately after the completion of injection is compression of cartoids on both sides; especially in the cyanotic group, as it is found that this concentrated dye in cerebral circulation damages the blood brain barrier. The other methods used have been direct heart puncture, retrograde through the subclavian route, and selective angiocardiography by cardiac catheter.

The technique of the investigation needs a preliminary film for check up for selection of operating factors of the X-ray Unit and also to test the serial changes which may sadly fail at the last moment as it sometimes does occur. The kilovoltage selected must give double the normal contrast, and the exposure factors should be about 1/400th of a second for sharper details. It is ideal to use a bi-plane angiocardiographic unit to minimise the dangers of repeated injectious to the patient, and it is an essential requirement in all modern radiological institutions. Simultaneous recording of the electro cardiogram gives the sequence of time and phasing of injection besides indicating conduction defects or ventricular arrhythmias ; the latter, the dreaded complication of the investigation, so that effective steps may be taken at its onset. Complete case records must be maintained before and after the investigation, and the investigation should not be repeated more than two injections twice. The dosage of the dye is calculated on the basis of the weight of the patient.

Under :

10 -	lbs.	0·75 - 1	c.c. per kilogram weight of the body.	
10 -	25 lbs.	0·67	c.c.	do.
30 -	60 lbs.	0·5	c.c.	do.
60 -	120 lbs.	0·38	c.c.	do.

Roughly 50 c.c. is the largest volume used, but if selective angiocardiography is done it is considerably less. In patients over the weight of 30 lbs. if the cardio thoracic ratio is over 60% an additional 20 - 25% of the dye is used. As a rough working measure, 1 c.c./kg. weight can be given. The injection should be given in 1 - 2 seconds, by air pressure on the piston of the syringe or by some mechanical contrivance. The aim of the investigation is to catch the first jet of injection which gives valuable information. Failure to do this will result in poor interpretation.

Space does not permit me to go into the details of the various types of camera units used with their advantages and disadvantages nor is there time to discuss the types of films used in these various units. In this Institute, there is single plane Elema Schonander Camera unit recently installed and brought into use creating greater interest in this field of diagnosis. Much is needed to bring this unit upto date, so that the thoracic surgeon gets all the data necessary prior to his operations, and proper interpretations could be arrived at. It is our fond hope that atleast the bi-plane angiocardiography unit with screening facility will come into existence soon giving the minimum requirements in this field of investigation.

Mention must be made of the reactions due to opaque media injected in a large volume as a bolus into the heart. Systemic reactions start as metallic taste in the mouth with intense heat spreading throughout the body ending in the lower and extremities last 10—20 seconds after the injection. This time factor is a great boon for the investigation is over by that time, so the patient is usually calm during the investigation.

There is a fall of blood pressure of about 30 mm. and a rise of pulse rate by about 30 more per second. Flushing of face and shoulders is common. Sweating and pallor are seen. Patients complain of nausea, weakness and dizziness. Occasionally urticaria or angioneurotic oedema occurs.

The reactions may be explained falling under various systems of the body. The cardio vascular symptoms such as arrhythmia during and after the investigation with depression of the ST segment or ventricular extrasystoles leading on to ventricular fibrillation and death are due to the acute coronary insufficiency caused by the non-oxygen containing contrast medium sent in by pressure injection as a bolus displacing blood. It may be also due to the chemical action of the dye on the myocardium or neurogenic reflex. The thrombosis of the vein in the arm is due to hypertonicity of the solution along with its chemical action.

There is an increase in the respiratory rate with a decrease in the tidal air and may result in apnoea ending in respiratory arrest and failure. This is due to chemical action on the respiratory centre or bronchial obstruction by angioneurotic oedema.

In the brain there is damage to the blood brain barrier after this investigation with even oedema and punctate haemorrhages. It is reversible unless severe and hence the precaution of pressure over the carotids immediately after the completion of the injection. This depends on the concentration and volume of the dye and the duration of it in the cerebral circulation. This is the cause of headache, dizziness, weakness, drowsiness, convulsions, syncope and confusion.

The gastro-intestinal symptoms are metallic taste in the mouth with nausea and vomiting. The cutaneous manifestations are flush, warmth, and urticaria and occasionally angio neurotic oedema. The miscellaneous reactions are, arm and shoulder pain and thrombosis of the vein.

Angio Cardiography

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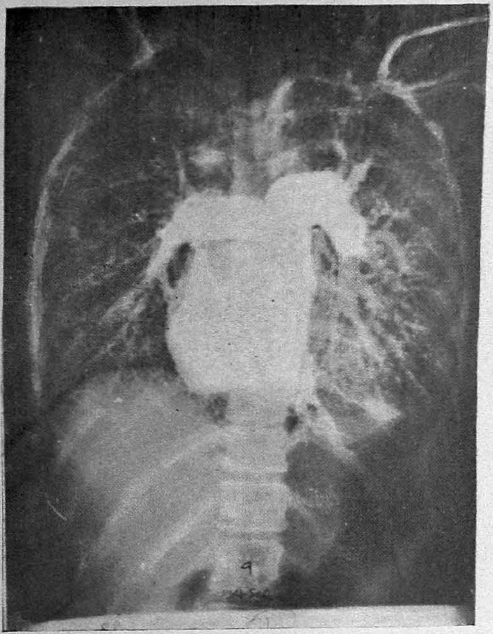
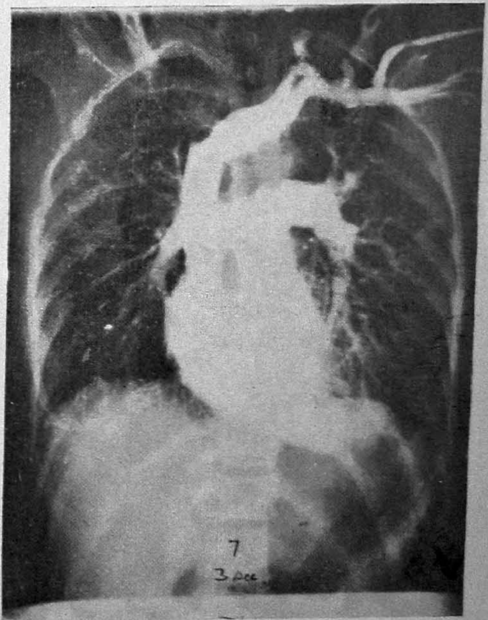
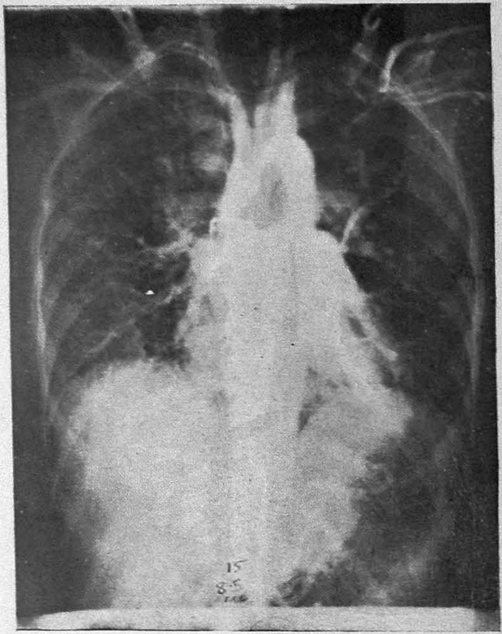


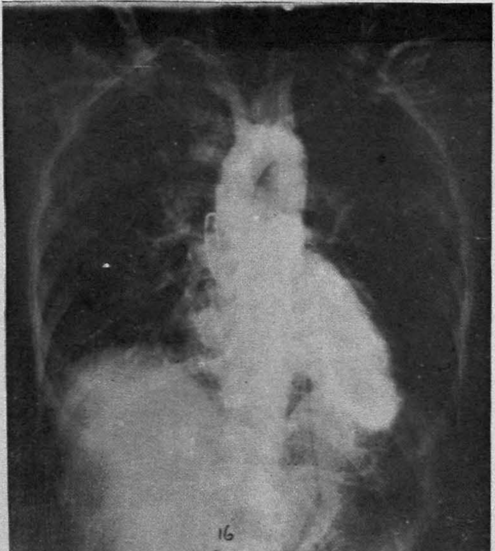
Right auricle, outflow tract of the right ventricle and pulmonary arteries.



Superior vena cava right auricle and right ventricle with pulmonary arteries.



Left auricle left ventricle in systole aorta and great vessels.



Left ventricle in diastole and aorta and great vessels.

The death in this investigation is due to:

1. Anaphylactoid reactions in 1-3% of cases.
2. Respiratory arrest.
3. Anuria.
4. Delayed reactions.
5. Cardiac arrest in cyanotic groups.

The factors implicated are:

1. Hypersensitiveness to iodine.
2. Chemical structure has been said as one of these but saline also can produce it and so this cannot be the cause.
3. Hypertonicity is not the cause for hypertonic solutions of glucose or saline do not kill the patient.
4. Impurities in solution.
5. Volume of the dose of the contrast medium or multiple injections.
6. *Speed of the injection.*
7. Premedication and anesthesia.
8. Cyanotic heart group.
9. General condition of the patient dealt under contra-indications.
10. Mongolism.

For correct interpretation one must have a knowledge of the developmental anatomy and also the normal position of the various chambers and the physiology of circulation. Certain normal events occur during the investigation which my be mistaken for the pathology unless one assesses this with the time factor of the cardiac cycle. A reflux of the medium from the right atrium into the inferior vena cava and hepatic veins during diastole is seen. It does not occur during systole as the caval orifices close during this phase. It is common to see this reflux during systole in pulmonary stenosis or arteriovenous shunts of

the heart. This reflux of the medium causes delayed opacification of the right atrium giving a suggestion of re-opacification through an atrial septal defect. The tricuspid valve is vertical in position and alters in position during the cardiac cycle, likely to be misinterpreted as Ebstein's anomaly unless the time factor and other co-existing conditions are considered together. The right auricle may assume 3 to 4 times the size in diastole compared with the systolic phase, not to be misinterpreted as dilated right auricle.

The right ventricle appears shaggy in appearance and contrasted with the right auricle shows as an U, the limbs varying in width with the age, the limbs being closer and narrower in old age. The conus and the pulmonary artery are separated by two sinuses appearing as bulges. The stenosis of the pulmonary artery may be of minor degree and easily missed; for stenosis need not be string like. The calibre of the pulmonary artery is equal to the aorta in infants and childhood and a little narrower than the aorta in adults. The pulmonary veins are lower than the hila and are seen as four in number crossing the pulmonary arteries at right angles. The pulmonary veins may vary in number and distributions. The left auricle appears as a quadrilateral chamber and may be seen on the left border in diastole. The left ventricle appears well in diastole otherwise it is shaggy in outline. Aortic valves may be seen as notches in the outflow tract. The ascending aorta is slightly wider than the descending aorta or the pulmonary artery in the adult. In cases of truncus it is twice the size of the descending aorta, the size being normal at the site of isthmus.

Having considered the normal appearances of the angio cardiogram, the abnormalities in the angiocardiogram can be studied. But space does not permit to go into the details of the various congenital lesions or even to consider the common anomalies. However, it is valuable to deliberate on the reading of the abnormalities of the angiogram. This necessitates step by step consideration of each and every shadow seen taking the sequence of time for their interpretations.

The abnormal sequence of opacification or re-opacification of chambers including premature staining of chambers of vessels means abnormal communications between the two circulations.

It follows therefore that lack of sequence or delayed opacification indicates an obstruction of the nature of stenosis or atresia.

Enlarged chambers are seen with obstructions or abnormal communications between both sides of the circulation.

Similarly the abnormal sizes of the vessels should be viewed. Larger pulmonary stem with narrower peripheral vessels are seen in pulmonary stenosis with post stenotic dilatation or high pulmonary hypertension. The peripheral vessels are large in arteriovenous shunts as in patent ductus arteriosus or atrial septal defect etc. It is common to see the left inferior branch of the pulmonary artery bigger in patent ductus arteriosus and the right pulmonary arteries larger in atrial septal defects. The right pulmonary artery is commonly smaller in congenital pulmonary artery aneurysm.

The presence of a left superior vena cava does not mean an abnormality unless there is anomalous drainage along with this as this vessel may drain into the left auricle. The pulmonary veins may drain into this. It may drain into the superior vena cava or right coronary sinus. Truncus communis is seen as a large vessel double the size of the descending aorta upto the isthmus where it becomes normal in calibre. In coarctation there is narrowing in this region of isthmus.

The altered contour of the chamber or the tracts is seen in cases of stenosis, aneurysms, and thrombi inside the chamber or vessel. Next the position should be considered like the position of valves as in Ebstein's anomaly. The abnormal vessels or aberrant origin of vessels and aberrant communications or drainage should engage the attention. The aberrant openings of the pulmonary vein are a common associated condition with other vascular anomalies. The flow and emptying of the dye will

reveal the form of stenosis or communications. Reduction in opacification is seen in shunts due to inrush of the fresh blood without contrast medium, even showing a filling defect.

Though this may sound quite easy of interpretation, time and again it is common to find difficulties to get a diagnosis without the aid of other investigations like the catheter studies with pressure readings and blood gas analysis, besides clinical and laboratory investigations. The same difficulties may not be experienced in selective angio cardiography. In quite a number of cases the lesions are multiple confusing the interpretation and vast experience is necessary to give a correct interpretation as this needs specialists' interpretation, instead of varied opinions from one and all. The Radiologist must have all the data for giving an expert opinion before the case is subjected to operation, on meagre guesswork. These problems in interpretations make the subject fascinating and interesting stimulating a keener study in this branch of medicine.

I thank the Dean, General Hospital, Madras and the Director, Institute of Radiology for permission to publish this.

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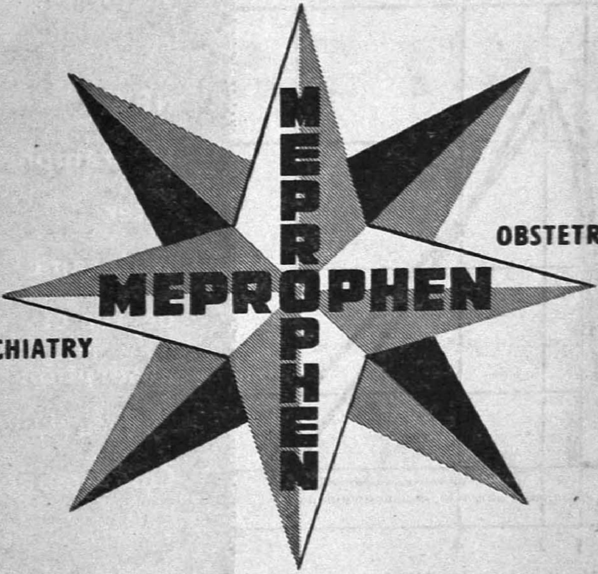

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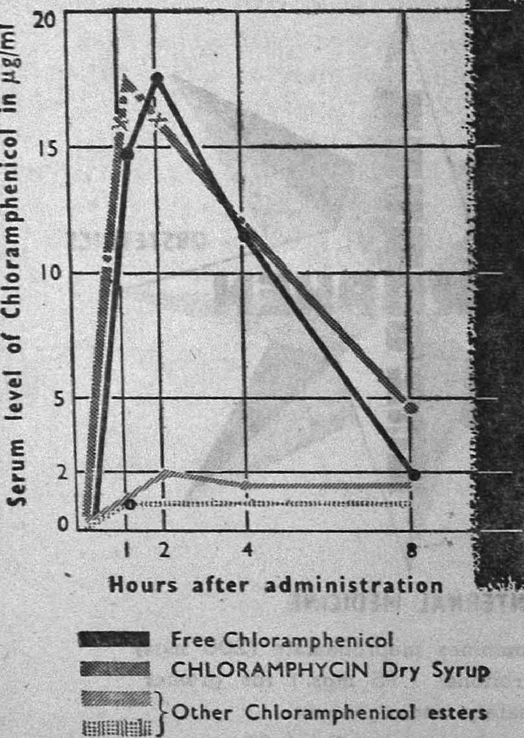

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Ossifying Haemangioma of the Muscle

Prof. S. P. VEDACHALAM
& Dr. S. ARUMUGAM*

Ossifying haemangioma is an uncommon tumour. It may arise *denovo* in the substance of a large skeletal muscle and not be associated with systemic or cutaneous angiomatosis.

Fulton and Sosman, in reviewing venous angiomas of striated muscle have reported 4 cases of phleboliths present in venous angiomas of the extremities. Angiomata may be congenital in origin but trauma plays a role either in attracting attention or making the swelling grow and become prominent. Pain of a paroxysmal or continuous nature may be present or alternate with increase in size. Turgescence or temporary enlargement of the lesion with an extremity, pendant or a tourniquet above has been noted. Radiographic evidence of calcification in areas where venous plexuses are not contemplated shows a possible clinical diagnosis. Aspiration biopsy taps blood only and no demonstrable tumour tissue. Usually the patient is well under 30 years. In long standing cases, ossification is possible (Pack & Ariel).

The tissues of mesodermal origin maintain a high degree of versatility and may with great ease develop into any tissue of such origin (Pack & Ariel). Ossification as part of intermutability in soft tissue growths has been recorded now and then. The mesenchymal cells have much wider potencies for differentiation —and particularly in abnormal conditions of growth wherein their normal potencies or plasticity is reverted. Proliferating mesenchymal tissue can produce all that embryonic mesenchyme

*Department of Pathology : Madras Medical College, Madras-3.

can produce. "The cells become less differentiated *de facto* as well as in appearance". This can only mean that their earlier potencies are reacquired. Most kinds of adult cell of mesenchymal origin can multiply, and when they do so, they often display their reacquisition of embryonic potencies by redifferentiating in aberrant directions. The young multiplying progeny of formerly differentiated cells probably possess pluripotency equivalent to that of undifferentiated mesenchymal cells which have never matured (Willis). More so this is the occurrence with vasoformative tumours as Willis records the occurrence of peculiar angiomatous structure in the muscles. Weiss and Williams described several cerebral cavernous angiomas which contained bone and glial tissue. Willis reports of a female 35 years, who died of haemorrhage in the anterior part of the right internal capsule. Histopathology revealed in this case calcified vessels. Wyn Williams reported a case of cerebral haemangioma, diagnosed incidentally at autopsy showing ossification. According to him this was the third cerebral haemangioma ossifying. Stout reports in one tumour composed of capillaries, adipose tissue and smooth muscle all present together.

We report here a case of ossifying haemangioma occurring in the deltoid muscle, for its rarity and for its unusual presentation. The following example is reviewed here for its rare occurrence as one of the ossifying haemangiomas in a rare site. The tumour was present for six years and has shown ossification probably with the advance of time.

Case Report.—Male K. aged 37 years was admitted into a surgical unit of the Government General Hospital on 29—9—'58.

Complaint.—Swelling of rt. upper arm 6 years with pain for the last 6 months. The swelling was progressively increasing. Dull Pain - 6 months. No other relevant history.

Local Examination.—A huge swelling, the size of a small melon, seen over the right upper arm extending from the

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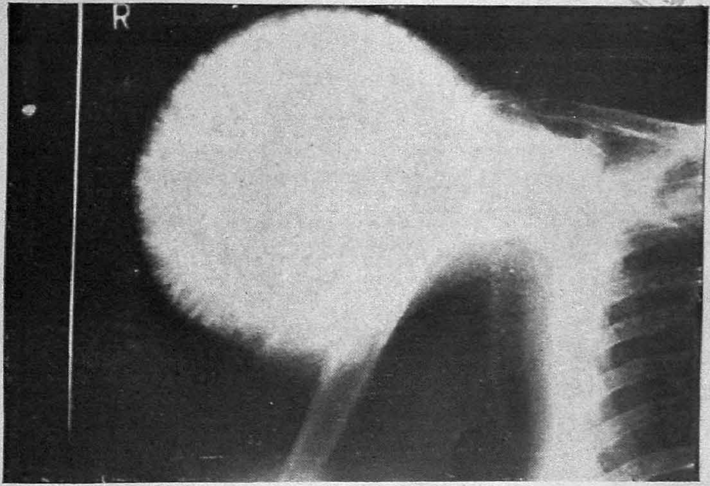

Fig. 1

X-Ray appearance of the Growth upper arm.

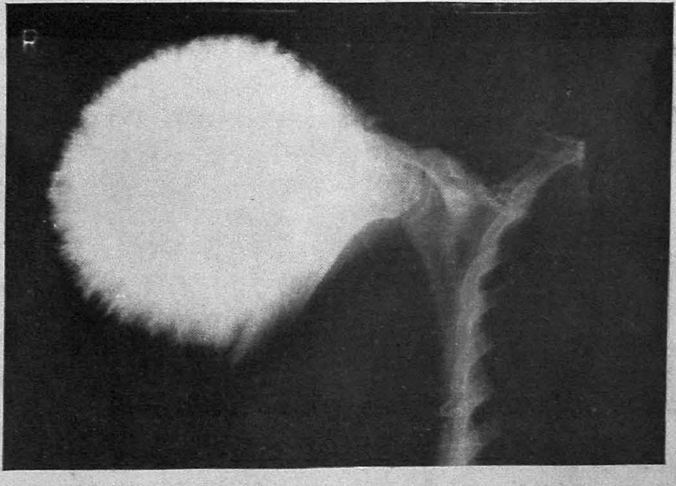

Fig. 2

This picture clearly shows the growth to be separate from the bone and limited to the soft tissues of the upper arm and also the joint to be free.

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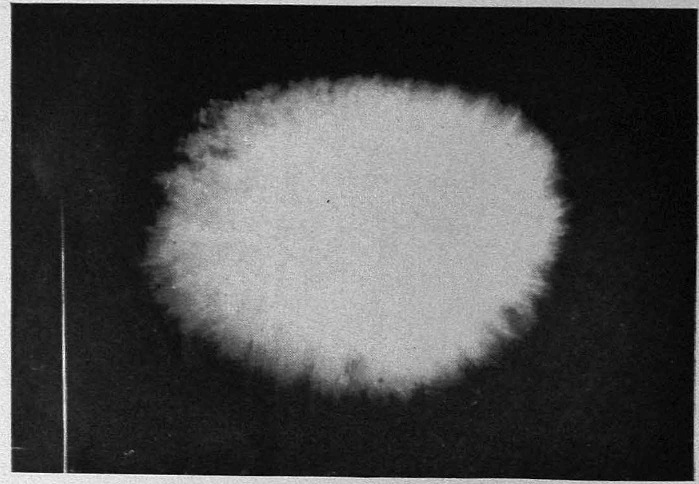

Fig. 3

X-Ray picture of the shelled out tumour.

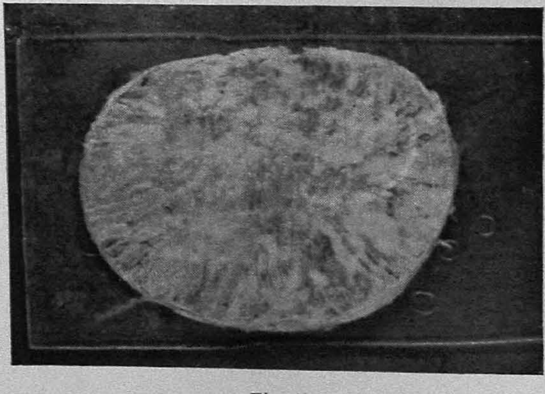

Fig. 4

Appearance of cut section of growth.

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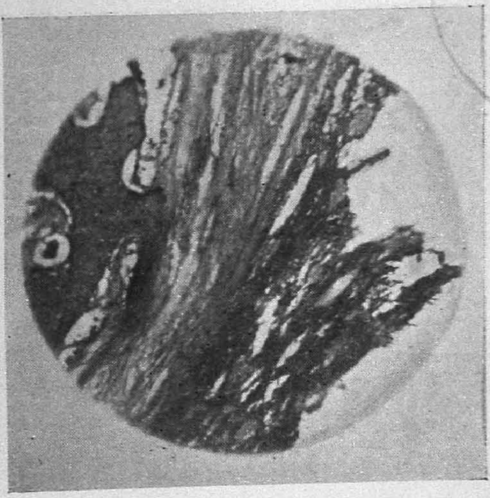

Fig. 5

Microscopic appearance which shows blood spaces and fibrous connective tissue showing ossification as strands

Fig. 6

Another area of the growth where cavernous spaces show ossification.

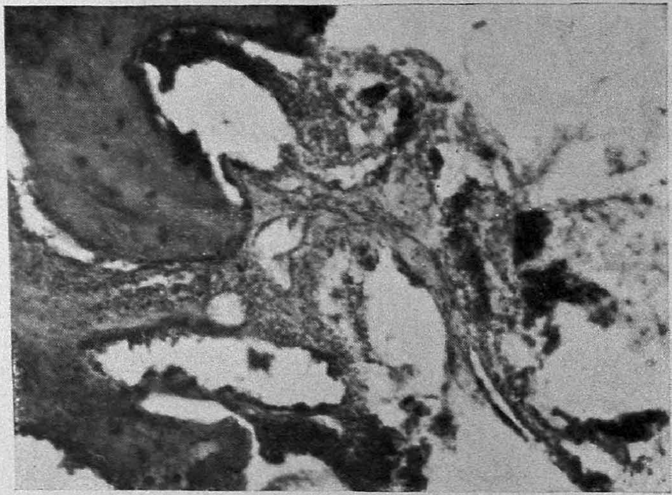

Fig. 7

Higher Magnification of Fig. 6.

acromion process to the middle of upper arm from above downwards and from midclavicular line medially to the middle of the back of forearm laterally. The swelling was warm to touch; slightly tender; surface bony, hard and irregular; margins definite. The swelling is movable and not fixed to the structures underneath. Skin over the surface is healthy. Few dilated veins are seen, not attached to the swelling.

General Condition.—Somewhat illnourished. Slightly anaemic.

X-Ray.—Shows a homogeneous radio-opaque mass circumscribed with spiky projections radiating from the central mass. An ossifying neoplasm in the region of deltoid—? Ossifying Haemangioma.

OPERATION NOTES

Under endotracheal anaesthesia, an incision in the deltopectoral groove was made. The tumour was found below the deltoid and pectoralis major; both the muscles were found stretched over the underlying tumour. The tumour *en masse* was enucleated. It was found that the tumour did not have any bony connection to the humerus. It had not infiltrated into the nerve or vessels. The tumour weighed 5 lbs., and was 6" in diameter, hard and could be cut only with a saw. Cut section showed that it is bony hard with radial striations. The wound was closed in layers.

PATHOLOGY REPORT

Gross.—As mentioned above, the growth had a bony-hard feel and presented a spiked appearance something like a pine-apple. The spikes mark the lines of ossification. The X-ray picture of the shalled out tumour is shown. It measures 6" in diameter and weighs 5 lbs. Rough and uneven surface and fairly compact, this neoplasm is separate from the underlying bone—[Pictures (i) a central slice & (ii) of the other two hemispherical ones.]

Micros.—The section shows venous spaces lined by calcified strands of connective tissue, most of which were ossified. The cavernous spaces contain blood.

Diagnosis.—Ossifying haemangioma arising from the deltoid muscle.

DISCUSSION

With the bony hard swelling, with dull aching and continuous pain, pain present only for the past 6 months, while the swelling had been there for the past 6 years and no trauma initially reported and no involvement of the joints and no other relevant findings—one should think in terms of neoplasm of the region. Since congenital, inflammatory, degenerative and traumatic conditions are ruled out by the history and clinical picture, we are left with a group of tumours from skin downward to the bone. But the points, that a few dilated vessels were seen coursing over the swelling and the swelling is freely movable and not fixed to the deeper structures, show the bone to be free; yet the swelling was bony hard. The spiculed projections in the form of spikes, revealed by the skiagram free from the bony margins of the humerus, support an ossifying growth apart from the bone outside in the soft tissue. Even though, periosteal soft tissue growths, as periosteal fibro sarcoma may simulate the appearance mentioned above, the clinical history, marked by a long duration of six years and the relative absence of pain and disability for a later part of the period, rules out such osteogenic growths of the periosteum. Also, the X-ray appearance precludes such a possibility. At the operation, the tumour was found to be free from the humerus. The tumour could be easily shelled out. At this stage, an insight into the nature of the growth was available to the Surgeon that it was not involving the bone. The intact periosteum and the integrity of the bone exclude all osteogenic tumours arising from the humerus. As tumours of the bone—should show continuity with the periosteum of the bone and either sclerotic or lytic changes, let alone a clear cystic area, the radiological appearance lacking their features is proof against a neoplasm arising from the bone (Geschikter & Copeland). The absence of infiltration into vessels and

nerves, thereby proves that it is not malignant. The histopathological appearance of venous spaces containing blood and showing connective tissue strands ossified and the ossification progressing along the strands of tissue which keep the tumour in position giving the sunray appearance is highly suggestive of an ossifying haemangioma arising in the substance of the muscle.

SUMMARY

A case of ossifying haemangioma arising from the deltoid muscle is reported in view of its uncommon occurrence and on account of the perplexity at the time of examination and its benignity.

Sincere thanks are due to the Surgeon in charge of the case and the Dean, Government General Hospital and Madras Medical College, Madras.

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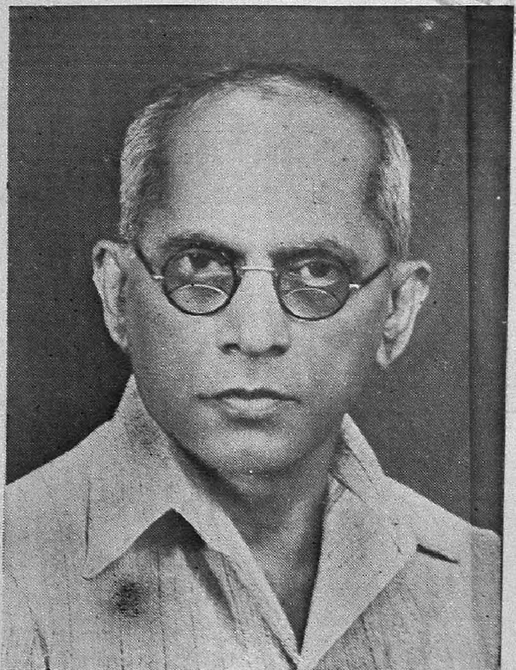

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once the Hon'ble the Minister of Public Health,
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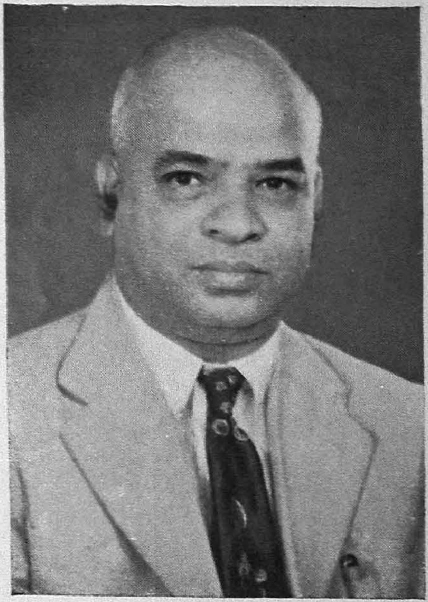
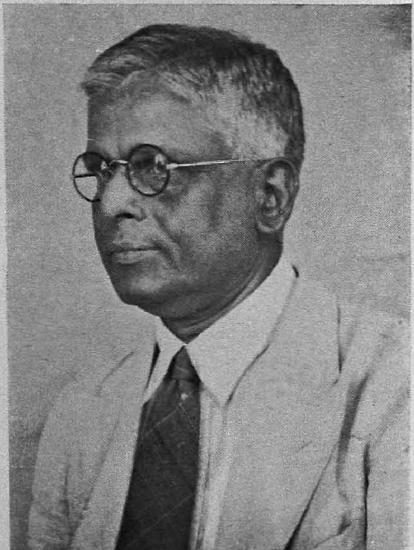


Late Dr. R. K. K. TAMPAN,
M.D., M.R.C.O.G.
Retired Director of
The Up-graded Institute of Obstetrics
and Gynaecology, Madras



Late Dr. A. SRINIVASALU NAIDU, M.D.
Retired Dean of
The College of Integrated Medicine,
Madras

IT IS WITH DEEP FEELINGS OF SYMPATHY THAT WE RECORD THE DEMISE OF:

Sri A. B. Shetty was the Minister for Public Health for nearly a decade in the different cabinets of the Madras State and Mysore State till just before his retirement from active politics. His versatility and unflagging interest in the subject of public health have been too well known to require a fresh reminder. How often was he mistaken for a medical graduate when he spoke lucidly on subjects allied to public health! The medical fraternity particularly those of the Madras Medical Service will ever remember him with gratitude. May His soul rest in peace!

Srimathi A. Parukuttiraman reached the high rungs of the ladder in the Madras Medical Service and retired as Civil Surgeon and Superintendent of the K. G. Hospital, Triplicane. The Madras Medical Service Association owes to her a deep debt of gratitude for her advice and guidance given in the capacity of the Vice President of the Association, a position that she held with dignity. Our sincere condolences to the relatives.

Sri A. Sreenivasulu Naidu, M.D. was a giant in his specialty. Who can forget the day when he proved formidable in many a nerve racking medico-legal sessions in the High Court of Madras convincing the court of the guilt of the murderers. As Professor of Jurisprudence and Police Surgeon and later as Dean of the College of Integrated Medicine, he acquitted himself with honour. He was Chairman of Dr. M. R. Guruswamy Mudaliar Library Committee in which capacity and similarly as an active member of the Madras Medical Service Association, his deliberations helped to guide the destinies of the Association in a respectable manner. His end was sudden due to a fatal accident on the famous highway of Madras. The loss is irrepairable and we sincerely condole with the members of the bereaved family.

Dr. K. Vasudeva Rao, M.D., F.R.C.P., was a notable member of the Service Association. He held the highest post as the Director of Medical Services in the State. In the field of Tuberculosis, he was an authority and to the patients stricken with Tuberculosis he acted the angel of relief. Highly qualified, his services rendered to the State will remain ever fresh. We offer our sincere condolences to the members of the bereaved family.

Dr. R. K. K. Tampan, M.D., F.R.C.O.G., was the first Director of the Institute of Obstetrics and Gynaecology and an active member of the Service Association. Full of activity and cheer, he passed away in harness. We offer our sincere condolences to his beloved kith and kin.

Srimathi R. Subramaniam has been the noble wife of the reputed Physician of the State, if not our country. Well known for her piety, benevolence and high culture, she not only played the role of the gentle wife but also that of the worthy mother. The sudden and premature demise of Srimathy Subramaniam has left a void in her home, difficult to fill. Our heartfelt sympathies are with Dr. R. Subramaniam, the Professor of Medicine, his beloved daughter and the son. She will be ever remembered for the selfless services she had rendered by the magnificent collections she had made towards the Cardiology Fund.

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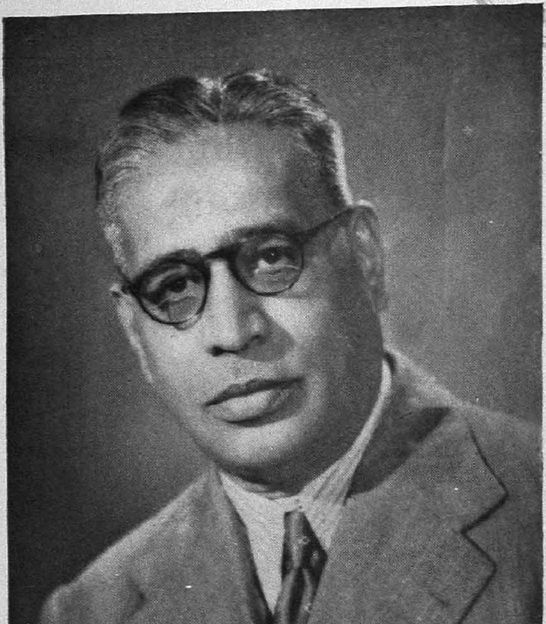

Late Dr. K. VASUDEVA RAO, M.D., F.R.C.P.
Retired Director of
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Late Srimathi PARUKUTTI AMMA
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Dr. R. SUBRAMANIAM, M.D., M.R.C.P. (Lond.)
President, Madras Medical Association.

MY COLLEAGUES AND FRIENDS,

We are happy to meet again at the end of the year to have our annual function. We are equally happy that Dr. Ananthanarayana Iyer has kindly consented to address us this evening. Before requesting him to do so, I should like to place before you, the Members of the Madras Medical Association, some of our problems which need to be considered.

The Magazine, as you know, continues to be irregular. This is because of the lack of interest evinced by the Members. I am willing to approach the various pharmaceutical concerns to give the required financial assistance so that we would not be dependant on the Association's resources alone, to meet the expenses connected with the Magazine. The mofussil members have to be informed of the current progress in the various aspects of medicine, surgery, midwifery and laboratory science, and it is upto us who are in the teaching institutions to guide them. If each one of us could put in a year's advance that we have gathered, it will go a long way to help the mofussil members to catch up with the progress.

In the Legislature, the medical officers have been held responsible for overcrowding in the hospitals. There is no denying the fact that the City State Hospitals are overcrowded incredibly. The hospitals were built at a time when the population of the City was only 2½ to 3 lakhs, but to-day the population is above 14 lakhs—in other words it has multiplied by roughly 4 to 5 times, of what it was. Along with the increase in population, the bed-strength should have been increased correspondingly and the hospitals provided with better facilities, equipment and better-trained personnel. On any O. P. day—I am willing to make a challenge—let the legislative members who cast aspersions against the profession, or for that matter, any member of the legislature or any independent private practitioner come forward and see

the O. P. strength. On an average we admit about 40 cases each day. In any western country only about 10 cases are admitted per day. 3-4 times the number of cases we admit here in India. It is not a pleasure to work in crowded wards. We are not paid one naya paisa extra for the additional labour. It is under great strain that the medical officers, their assistants, the house staff and more particularly the nursing staff and the menial nursing staff work. I may be permitted to point out that this *terrific* strain on the medical staff cannot go on indefinitely. They are bound to break down.

A harbour cooly patient in my ward pointed out to me one day that the hospital menial staff are working on a pay which is far below his own earnings; besides, their work is easily 10 times more strenuous. He would certainly not exchange his position even with that of the ward boy. While people here work under this terrific strain, a member of the legislature made the allegation that the medical officers admit cases to earn cheap popularity. It is unfortunate that the medical officers cannot reply to the members of the legislature. It is unfair to attack a person who cannot reply. Through this General Body, I request the Minister of Health, who unfortunately is not here, to safeguard the prestige of the service members. If he allows a member of the legislative council to criticise service members unnecessarily at a time when they are working hard on ill-paid wages, it will only be courting disaster. Unlike the other sections of society, we do not demand that we will work only for a stipulated time and that we will look after only the sanctioned strength of beds.

In season and out of season from the top man in the country to the third-rate politician, they are advocating medical men should go and serve the villagers. Modern techniques in medicine do not facilitate practice at the village level. Even in the most advanced countries like the U. S. A., every village does not have its doctor. When that is so, how could a country like ours which is economically very poor and where doctors are hopelessly small in number achieve it overnight? It is nothing more than mere lip sympathy to the suffering villager when doctors are requested to come and settle in villages. The solution to this problem of the villagers will be to develop mofussil hospitals, *i.e.* District and Taluq Headquarter Hospitals by placing an ambulance at the service of these hospitals. A minimum of six doctors must be made available for each Taluq hospital, so that when an emergency call comes, they should be able to spare a doctor to attend to the emergency, and if the patient requires hospitalisation, he may be brought by the same ambulance.

The doctors in the villages are not provided even with the ordinary amenities. Many dispensaries in the *mofussil* do not have essential drugs. A list of essential drugs which are not available has been drawn already. After

all a doctor is also a human being and he needs some rest and in the *mofussil* hospitals prohibition cases are brought in at all odd hours and the doctor's services are required. It is one of the most unpleasant tasks for the doctor to attend to prohibition cases day in and day out. At the end of a few months he feels he should be much better-off in his hometown. With the meagre pay the Government gives, he has to bother very much about the prohibition cases. What is worse is, the prohibition cases are not always genuine. If he is a young medical man and has sufficient courage to say 'No' to what the police demands, thereafter even with his courage, his life in the village is unpleasant. If the villager who is brought for declaration, happens to be an important man in the village, he threatens the doctor with physical violence and on more than one occasion, an Assistant Surgeon had to run up to me for advice as to what he should do—whether he should stick to the village and face the consequences of being treated badly or run away from the station. I, for one, am not averse to offer a solution to this unpleasant situation. In the dispensaries where the shelves are practically empty and no instruments are available even to carry out very minor surgical work, is it any wonder that our young men find it trying to work in the villages? Even boys and girls who are very enthusiastic, once they go to these places and come to grip with realities are bitterly disillusioned since they cannot make full use of what was taught to them in the Colleges. Another problem arising from taking up work in rural areas is that medical practitioners with families are unable to educate the children in the villages. They are obliged to maintain a separate establishment in a Taluq or District Headquarters or some of them, in the City itself. The result is they have to run two establishments. In some cases these people have to support their aged parents at home. That means they have to maintain three separate establishments.

In many of the mofussil towns competition is keen between the ordinary private practitioners who do good work and these hospital assistant surgeons. The result is, the practice is almost nil and the doctors feel they would be much better off in their own hometowns where even if they earn half or 1/3rd of what the Government pays, they will be called upon to do much less work and at more convenient hours. While these people do not complain about the labour cases, they complain much about the prohibition and medico-legal cases. Sometimes the young man has got to go 10 or 15 miles to see a body which is in a highly putrified state and offer his remarks.

Many of these men join the service with the hope that they will be given some sort of opportunity for higher studies. I was told that in the selection of the diploma courses, Diploma in Ophthalmology, E.N.T., D.G.O. and D.A. held recently, very few service candidates were chosen.

I do not know why when our boys and girls are available, they were not selected. Whatever may be the reason I may straight away say this is likely to demoralise the young men and women and produce a feeling that their service is not being recognised. I sincerely appeal to the authorities in power to look to this aspect of the problem, and encourage our boys and girls to study at least for such higher qualifications which the University permits.

In the last few years the asstistant surgeons who sit for higher examinations are given Rs. 100/- p.m. as a stipend. While I consider the Rs. 100/- much better than no remuneration, I feel this is very inadequate. After all a boy who is appearing for his M.D. or M.S. examination has to put in more work and study hard. His knowledge is utilised for the benefit of the people of the State. When that is so, I think that they should be paid at least their basic pay during the period of training (particularly when we do not pay any sort of higher remuneration for those who have higher qualifications). These are some of the thoughts that crossed my mind and I place them before you and I intend placing them before our Health Minister through you for speedy consideration.

Minutes of the General Body Meeting of the Madras Medical Association held on 5—10—1959 at the Madras Medical College, Madras.

The General Body Meeting of the above Association which was held on 5-10-1959 at the Madras Medical College was attended by 62 members.

Dr. R. Subramaniam, President of the Association in his opening address said that the Annual report had already been circulated to all the members and as there were no objections, it can be taken as passed. Dr. K. P. Sarathy seconded it. He also said that before proceeding further, a resolution might be passed on the untimely death of Dr. A. Srinivasulu Naidu and Dr. R. K. K. Tampan both of them being distinguished members of the Madras Medical Service. He requested all the members to stand for a few minutes as a mark of respect.

The following resolutions were passed by the General Body:

"This Meeting resolves to place on record the deep sense of grief at the tragic death of Dr. A. Srinivasulu Naidu, an old and distinguished member of the Madras Medical Service and convey their condolence to the members of the family".

"This meeting places on record the deep sorrow at the death of Dr. R. K. K. Tampan, a distinguished member of the Madras Medical Service and convey their condolence to the members of the bereaved family".

The above two resolutions were decided to be communicated to the members of the bereaved family.

Dr. T. Janardhanan then said that before they begin their routine work, they should also condole the death of the President's wife. He said that though it is not an occasion to upset him (President) anyway, the feelings of all the members of the service are equally with him in that sad demise which was unexpected. He said that he wished to convey on behalf of the Association their condolence on the sad demise.

Dr. Janardhanan then proceeded to inform the members that this year was an unusual one in which the audited accounts of the Association is not presented. He said that this is due to our ignorance of the new Companies Act. He stated that the accounts were sent for auditing to the newly elected Auditor Mr. Gopalakrishnan. He said that the auditor was not aware of the objection till he went through the new Companies Act, and stated that the election of the auditor for the year is not valid. The Secretary then read a draft letter to be sent to the Central Government and said that subsequent to the reply from the Government of India, an auditor can be appointed. He also pointed out that we will have to give a notice to the retiring auditor when he ceased to be the auditor of the Association. He said that a new auditor should be proposed and his name circulated to all members for approval by the General Body.

Dr. M. Natarajan asked why this Association was registered under the Companies Act and that whether it could not be registered as a private society.

Dr. R. Subramaniam said that but for the registration of the Association under the Companies Act, the meeting would not have been held to-day. He said that by registering as a Company, we are forced to do certain things. Otherwise, there will be a tendency to take things easy.

Dr. P. S. Kumaravelu wanted to know why the new auditor should not go through the accounts atleast.

Dr. T. Janardhanan replied that the only thing in the accounts is the large arrears of subscription (Laughter). He also informed that the Executive Committee has passed a resolution that the names of members who have not paid subscriptions consecutively for the last 3 years will be removed from the rolls.

Dr. R. Subramaniam then pointed out that practically all the requests of the Association to the Government have been negative and said that nothing prevents them from reiterating the resolutions.

Election of Office-Bearers :— Nominations in respect of President, Vice-President (City) and Executive Members have been received and nominations have been found to be in order. As there were no nominations, the nominated members were taken as elected unanimously. But in respect of mofussil Vice-President, there is one vacancy.

Election :—Dr. R. Subramaniam stated that there is one vacancy for mofussil Vice-Presidentship. He said that Dr. T. V. K. Iyengar's name has been proposed. Dr. S. Govindarajan seconded the name of Dr. T. V. K. Iyengar as one of the Vice-Presidents.

The President then informed the members that the nomination of the Auditor has to be postponed to another General Body which will be held sometime in December.

The President said that in view of the large arrears of subscription, he would suggest that if members pay Rs. 100/-, they become life members which will also save a lot of correspondence between the Secretary and members. There was a discussion on the above subject among the members and the following resolution was proposed by Dr. A. L. Annamalai and seconded by Dr. M. Natarajan.

" A fee of Rs. 75/- for Life Membership in one instalment or in two instalments of Rs. 40/- each to be paid within a calendar year. This will also apply to existing member ".

The President promised to become the first life member.

Dr. T. Janardhanan then read out a letter of the Chairman, of the Pay Commission to the members. He also read the reply sent to the Pay Commission to the above letter. He also stated that the Commission may call us for an interview and wanted to know whether there were any suggestions from the General Body. There were no suggestions except to increase the annual limit of pension from Rs. 5,000/- to Rs. 6,000/- and a resolution was passed to increase the annual maximum limit of Pension from Rs. 5000/- to Rs. 6,000/-.

Then Dr. K. G. Menon put forward 3 resolutions to be passed by the General Body. The first one was regarding Selection Grade Assistant Surgeons. After some discussion, the following resolution was passed by the General Body. Dr. P. D. Kannivelu seconded the resolution.

" In the matter of the Selection Grade Assistant Surgeons those whose claim has been overlooked because they are in the teaching line or because

they are in the special departments, they may be made super-numerary Selection Grade Assistant Surgeons with the same grades of pay"

Dr. T. Janardhanan then read out a resolution received from Dr. T. Raghavan which runs as follows:

"It is observed that in the matter of promotion as Selection Grade Assistant Surgeons, the seniors are skipped over and the juniors are promoted just because the seniors happened to be in the special department and or as Tutors. This is an anamolous position requiring correction. Hence it is requested that whenever a junior person is promoted as Selection Grade Assistant Surgeon the senior may be given supernumerary post as in the case of Civil Surgeons".

There was a discussion on the above resolution. Dr. S. Govindarajan said that the supernumerary Civil Assistant Surgeons be created just like in the case of Civil Surgeons. Dr. K. G. Menon said that the people from general line are posted to T. B. Sanatorium and persons with specialist qualification are not posted. The above resolution was passed.

Dr. R. Subramaniam said that last year a resolution was passed that 20% of the Civil Assistant Surgeons should be made Selection Grade Civil Assistant Surgeons. He said that before our suggestions were sent, the Director of Medical Services himself has recommended the same to Government and it is pending with the Government.

Dr. K. G. Menon said that it is the feeling of all the Assistant Surgeons that the Association is not helping them and is not taking interest in them.

Dr. R. Subramaniam replied that the responsibility is only with the Assistant Surgeons. He said there are 1,400 Assistant Surgeons but the members are only 400. He said that again and again the District Medical Officers were requested to enrol all the Assistant Surgeons as well as themselves as members of the Association. He said that they did not join and if they had increased the strength of the Association, our voice will be heard or otherwise the Association will die out.

Dr. S. Govindarajan pointed out that he is a member of the Association for a long time and as the President had said it is fault of the members of the Service if their claims are not redressed. He also said that a promotion has to be effected taking into consideration the confidential report also of the officers which are confidential between the D.M.S. and the Government and only matters of principle can be raised by the Executive

Committee. He said that the Executive Committee has taken matters of principle and passed many resolutions. It is not the fault of the Executive Committee or the Association but it is their bad luck that they do not have sufficient members on the rolls. He said that in so far as the recruitment drive is concerned the President of the Association has requested all the District Medical Officers to take greater interest in our Association. You may note no District Medical Officer is present today here, he said.

Dr. T. Janardhanan said that when the District Medical Officers Conference was held in the City, the District Medical Officers were requested to try to enrol as many members as possible for the Association. He said that all methods were tried and the only way now is to form a separate recruitment Committee.

Dr. V. Rajagopalan suggested that when persons are recruitted as Assistant Surgeons, an appeal should be sent to them to join the Association.

Then the President requested the General Body to select persons for the Recruitment Committee. The General Body selected the following persons for the Recruitment Committee :

City :

1. Dr. Arthur Daniel.
2. Dr. P. S. Kumaravel.
3. Dr. V. Rajagopalan.

Mofussil :

1. Dr. Khaleelur Rahman, *D.M.O., South Arcot.*
2. Dr. M. V. Krishnamurthy, *D.M.O., Coimbatore.*

The above members have been given power to co-opt by the General Body.

Dr. T. Janardhanan then read a resolution received from Dr. S. Ranganathan which runs as follows. It was seconded by Dr. S. Govindarajan.

" The number of Civil Surgeons" posts in the Madras Medical Service is very few taking into consideration the strength of Assistant Surgeons and Selection Grade Assistant Surgeons. It is suggested that the number may be increased by promoting all the Assistant Surgeons holding charge of a hospital whose bed strength is 100 and above and post of R.M.O., T.B. Sanatorium, R.M.O., Erskine Hospital, Royapettah Hospital, Government Ophthalmic Hospital and other big hospitals.

The General Body discussed the above resolution and it was decided that Hospitals like T.B. Sanatorium, Erskine Hospital, Hospital of Integrated Medicine, Women & Children Hospital, Royapettah Hospital, Government Ophthalmic Hospital and R.M.Os. in certain big hospitals may be included to give effect to the above resolution.

Dr. M. Govindarajulu proposed the following resolution which was seconded by Dr. M. Natarajan.

"Many members of the service are now posted as Lecturers and Tutors in the Government College of Integrated Medicine. As this teaching experience is not recognised by the University or the Government it affects the prospects of many members of the service".

The General Body decided to withdraw the above resolution after some discussion.

The President then closed the meeting.

R. Subramaniam,
President.

T. Janardhanan,
Hony. Secretary.

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CURRENT REVIEW (DERMATOLOGY)

ADVANCES IN THERAPY

1. Chelating agent in seleroderma: The use of Edathamil has been commented on by Sigfrid A. Muller, Louis A. Brunsting and R. K. Winkelman in A. M. A. Archives of Dermatology August 1959. Reports are favourable and the drug used intraveneously appears to be of some benefit.

2. Relaxin in ulcers and Gangrene of collagen disorders - Hugh Reynolds and Clarence S. Livingood: in A. M. A. Archives of Dermatology - October 1959. 1 c.c. each day in combination with steroids.

3. Tirquin (Triple synthetic Anti-malarial) (Aralen, Plaquenil and Atabrine) - has been tried in L. E. discoid and light sensitivity eruptions: Results compare favourably with other antimalarials - James H. Bennet & Rees. B. Rees in A. M. A. Archives of Dermatology - September 1959.

4. Topical Amphotericin - useful as an Antifungal Antibiotic in moniliasis - A. M. A. Archives of Dermatology - October 1959.

SUBJECTS OF TOPICAL INTEREST

1. Bowen's Disease and Systemic cancer. The association of the cutaneous with internal neoplasms is of interest - in respiratory, g. i. tract, genito urinary, r. e. system, skin and breast. - report by James H. Graham & Elson B. Helwig in A. M. A. Archives of Dermatology - August 1959.

2. Association of sclerosing haemangiomas with overlying epidermal neoplasm (Basal Cell) is reported in 25% of cases - Hilland J. Halprya and Arthur C. Allen - A. M. A. Archives of Dermatology - August 1959.

3. Urticaria Pigmentosa and associated general flushing due to 5 H. T. and its detection in urine is reported by AR. Brit. and M. Nickersen in A. M. A. Archives of Dermatology - August 1959.

4. The concept of Dariers disease as being neoplastic is put forward by Shelly et al in A. M. A. Archives of Dermatology - September 1959.